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Original Communications

A CONTROL GROUP FOR STUDYING THE END-RESULTS OF THORACOPLASTY*

AN ANALYSIS OF THE COURSE OF THOSE PATIENTS REFUSING OPERATION

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THE significance of this report lies in the fact that it tests the results of a therapeutic procedure against a natural control group. The real proof of the efficacy of therapy is found not alone in the analysis of a series of treated cases, but in comparing the fate of the treated cases with that of the untreated cases. It is well known that the opportunity to do this arises infrequently in clinical medicine, usually because of the difficulty in tracing the untreated cases. In tuberculosis such a study is of particular importance, since disagreement still exists in the medical literature concerning the fate of patients with pulmonary cavities.

In Cleveland, 750 tuberculous patients are in sanatoriums under the medical supervision of one group of physicians. All recommendations for surgical procedures are made in conference, and the operations are performed by one team of surgeons. Between 1932 and 1934, inclusive, thoracoplasty was recommended for 153 patients (excluding those with tuberculous empyema). Of this number 85 accepted operation, and 58 refused. Ten patients accepted operation one year or more after an initial refusal, and will be discussed separately. During the first three months of 1936, we were able to determine the condition of 114 of the 125 surviving patients in the entire group. With a few exceptions all

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The patients studied in this report are from the services of the Lowman Memorial Pavilion of Cleveland City Hospital, the Sunny Acres Sanatorium, and the Mount Royal Sanatorium.

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of these individuals were seen personally, the study including physical examination, roentgenograms of the chest, and examination of the sputum. The follow-up study, however, was not confined solely to this single examination, since the majority of the survivors have been followed for a period of one to four years in the sanatoriums, or in one of the clinics associated with them.

A study comparing the results of thoracoplasty with the fate of a group of patients refusing operation avoids certain of the criticisms which can be directed at the usual presentation of general thoracoplasty statistics. The recommendations for operation in our patients were made with no knowledge as to whether the patient would refuse or accept; hence the same criteria were employed in all cases. The analysis of the results, therefore, has a distinct advantage over the attempts at therapy evaluation arrived at by comparing the statistics of one sanatorium with those of another, in which different criteria for therapy may have existed; in which varying numbers of good and poor risks may have been operated upon; and in which differences in operative technique may have been employed.

The group here presented is entirely unselected, representing as it does all the different types of patient to be found in the general population of a large city. The statistical results, therefore, are not affected by the important selective factors of the patient's intelligence and economic status, which may influence the results in the case of studies made in health resorts or in individual institutions. The same facilities for sanatorium care and medical supervision were available to every patient in both the thoracoplasty and the refusal groups.

Finally, this study is not concerned with any comparison of methods of collapse other than thoracoplasty, since in both the operated and the refusal groups all the collapse procedures indicated were employed before thoracoplasty was recommended.

For simplicity of presentation the present status of the thoracoplasty group and the control group consisting of those who refused operation will be compared in two particulars:

1. The physical status: the effect of therapy in producing healing of the tuberculous disease.

2. The functional status: the effect of therapy in restoring the patient's ability to work.

PHYSICAL STATUS

Because of a complete lack of uniformity in the use of terms employed by various writers in describing the end-results of thoracoplasty, an exact definition of the terms employed herein is essential.

Closed.—Those patients are considered "closed" whose sputum is persistently negative for tubercle bacilli (including examination after concentration); whose roentgenograms are consistent with a healed or

retrogressive lesion without evidence of cavitation; and who show no evidence of constitutional symptoms. In the case of the thoracoplasty group, considerable reliance must be placed upon sputum examination, since the roentgenogram may be misleading with respect to the presence of cavity even with the use of heavy exposures with the Bucky technique. All patients included in this group have had negative sputum over periods varying from six months to three or more years.

Improved.—Those patients are considered "improved" who present only an occasional positive sputum with few bacilli; whose roentgenograms show a retrogressive lesion with conspicuous reduction in the extent of cavitation; and who show no evidence of constitutional symptoms.

Unchanged.—Those patients are considered "unchanged" who present persistently positive sputum; whose roentgenograms show a stationary lesion without conspicuous change in the extent of cavitation; and who remain with no change in the degree of their constitutional symptoms.

Worse.—Those patients are considered "worse" who present persistently positive sputum; whose roentgenograms show progressive disease; and whose constitutional symptoms are increased.

Dead.—Included under this heading are all deaths occurring after thoracoplasty was recommended.

Following the suggestion of Archibald, several authors within recent years have pointed out that the presentation of general statistics on thoracoplasty may be misleading; that for a more accurate evaluation of prognosis patients should be divided into classes based essentially upon a qualitative, rather than a quantitative, estimation of their disease. The reasons for this point of view will be made clear in the succeeding discussion. Such a classification has been proposed through the use of the aptly descriptive terms "good," "slipping" and "bad chronics."

Good Chronics.—In defining the "good chronic" we have employed the essential criteria recently enumerated by Brown and Sampson.[1] The patient has a cavity two centimeters or greater in diameter. The general condition is good, and over an observation period of several months, the patient has a normal temperature and pulse, a good appetite, no significant loss of weight, and is able to take some exercise. Expectoration and a positive sputum may be present. One further criterion is of importance. During the observation period the roentgenogram should show no evidence of any significant increase in the extent of the disease or in the size of cavities. It is obvious that regardless of his general condition any patient who shows roentgenologic evidence of progression of his pulmonary lesion cannot be labeled a "*good* chronic." In our series,

therefore, this group consisted of the individuals with chronic proliferative disease with cavitation, or with retrogressive caseous-pneumonic disease with cavitation.

Slipping Chronics.—All other patients in our series have been considered as "slipping chronics." In this group will be found the patients with constitutional symptoms, particularly moderate elevation of temperature, loss of weight, weakness, easy fatigue, and inability to take exercise. It also includes those patients showing, during the period of observation, roentgenologic evidence of progression of their disease. The group consists essentially, then, of the patients with progressive caseous-pneumonic disease with cavitation, and those with chronic proliferative disease with cavitation and recent exudative extensions.

The term "poor chronic" has been avoided, since but nine patients in the entire group of thoracoplasty recommendations during the three years under consideration, could clearly be placed in such a classification. The number was felt to be too small to warrant separate classification.

TABLE I

PRESENT STATUS OF THE COMPLETE THORACOPLASTY AND REFUSAL GROUPS

	THORACOPLASTIES 85 PATIENTS			REFUSALS 58 PATIENTS		
	NO.	%	%	NO.	%	%
Closed	48	57	66	6	10	17
Improved	8	9		4	7	
Unchanged	5	6	6	8	14	14
Worse	6	7	21	20	35	61
Dead	12	14		15	26	
Untraced	6[1]	7		5[2]	9	

[1]All six patients known to be living, but not available for recent follow-up study.
[2]Three of these patients known to be living, but not available for recent follow-up study.

RESULTS

Results in General.—Table I* shows the present physical status of the complete thoracoplasty and control groups. The condition of only 14 per cent of the control group remained unchanged. The course of the majority of these individuals who refused thoracoplasty was unfavorable, 35 per cent being worse within one to four years and 26 per cent being dead. By contrast, only 7 per cent of the thoracoplasty group were worse following operation, and the mortality rate of 14 per cent was scarcely more than half that of the control group. There were only 10 per cent (6 patients) in the control group who succeeded in becoming "closed" without operation, in contrast to the 57 per cent (48 patients) who became "closed" after thoracoplasty.

These six individuals in the control group are worth special comment. In only one case was cavitation greater than four centimeters. In four

*In the tables, fractions of a per cent have been eliminated, and the percentage raised or lowered to the nearest whole number.

instances, the cavities were extreme apical, and in the remaining two instances, subapical. The first patient, at the time thoracoplasty was recommended, had a five and one-half centimeter cavity in the left apex, with an ineffective pneumothorax of six months' duration. Operation was refused. The pneumothorax was maintained and the cavity became closed two years after the original thoracoplasty recommendation was made. The second patient had a four centimeter cavity at the left apex, with an ineffective pneumothorax of eleven months' duration. The pneumothorax was maintained and the cavity became closed one and one-half years after the refusal of thoracoplasty. The third patient also had a four centimeter cavity in the left apex. It was thick-walled and was known to have existed unchanged for one year prior to her admission to the sanatorium. Pneumothorax failed and thoracoplasty was advised and refused. A temporary phrenic nerve paralysis done elsewhere resulted in cavity closure about one year later. In the fourth and fifth patients, pneumothorax plus phrenic nerve paralysis had failed to bring about cavity closure after eight months and five months, respectively.

TABLE II

PRESENT STATUS OF THE "SLIPPING CHRONIC" GROUP

	THORACOPLASTIES 42 PATIENTS			REFUSALS 26 PATIENTS		
	NO.	%	%	NO.	%	%
Closed	18	43	57	1	4	8
Improved	6	14		1	4	
Unchanged	3	7	7	1	4	4
Worse	5	12	29	11	42	77
Dead	7	17		9	35	
Untraced	3	7		3	12	

At the time thoracoplasty was recommended the cavities measured three centimeters in diameter. The cavities unexpectedly became closed eight months and one and one-half years, respectively, after operation was advised. In the sixth patient a persistent three centimeter cavity, which had failed to respond to phrenic nerve paralysis after pneumothorax failure, became closed one year after the initial thoracoplasty recommendation.

In summary, it will be noted that after one to four years, 66 per cent of the thoracoplasty group were closed or improved, in contrast to 17 per cent of the control group. On the other hand, while 21 per cent of the thoracoplasty group were worse or dead, this was the fate of 61 per cent of the control group. In our series, therefore, *thoracoplasty increased by four times the chance of therapeutic improvement, and decreased by two-thirds the possibility of becoming worse.*

Results in the Slipping Chronic Group.—Table II shows the present physical status of the slipping chronic group. Of greatest significance is the fact that the course of most of these individuals who refused

thoracoplasty was unfavorable, 42 per cent being worse and 35 per cent dead within the one-to-four-year period. Only 12 per cent of the patients operated upon were worse after operation, and the mortality rate of 17 per cent was exactly half that of the control group. There were only 4 per cent (1 patient) of those individuals refusing surgery whose cavities became closed, in contrast to the 43 per cent (18 patients) whose cavities became closed with thoracoplasty. The one patient in the control group whose cavity became closed is the individual with the five and one-half centimeter cavity described above.

In summary, it will be noted that 57 per cent of the patients operated upon became closed or improved, in contrast to 8 per cent of the control group. On the other hand, although 29 per cent of the patients operated upon were worse within four years, or dead, this was the fate of 77 per cent of the slipping chronics who refused surgery. In the slipping chronic group, therefore, *thoracoplasty increased by seven times the possibility of therapeutic improvement, and decreased by slightly less than two-thirds the possibility of becoming worse.*

TABLE III

PRESENT STATUS OF THE "GOOD CHRONIC" GROUP

	THORACOPLASTIES 43 PATIENTS			REFUSALS 32 PATIENTS		
	NO.	%	%	NO.	%	%
Closed	30	70	75	5	16	25
Improved	2	5		3	9	
Unchanged	2	5	5	7	22	22
Worse	1	2	14	9	28	47
Dead	5	12		6	19	
Untraced	3	7		2	6	

Results in the Good Chronic Group.—Table III shows the present physical status of the good chronic group. The condition of 22 per cent of the control group remained unchanged. In this group of patients, however, the most favorable from the prognostic point of view, 28 per cent were worse within four years after surgery was refused, and 19 per cent were dead. By contrast, of those good chronics operated upon, only 2 per cent were worse, and the mortality rate of 12 per cent was lower by one-third than the corresponding rate in the control group. Of the patients who refused operation 16 per cent (5 patients) became "closed." In the case of the good chronics, however, thoracoplasty produced the most satisfactory results, since 70 per cent (30 patients) became "closed" by operation.

In summary, it will be noted that 75 per cent of the patients operated upon were closed or improved, in contrast to 25 per cent of the control group. On the other hand, although 14 per cent of the patients operated upon were worse or dead, this was the fate of 47 per cent of the control group. In the good chronic group, therefore, *thoracoplasty in-*

creased by three times the chance of therapeutic improvement, and decreased by slightly more than two-thirds the possibility of becoming worse.

DISCUSSION

It is apparent from Table I that among patients for whom thoracoplasty seems indicated, the cavities may become closed without operation in a few fortunate individuals. Of greater significance to the patient for whom thoracoplasty is indicated, is not the fact that in this series 10 per cent recovered without operation, but that within the short period of four years, 61 per cent of those patients not operated upon were either worse or dead. A comparison of the fate of a control group such as we have presented with the outcome in a group of patients receiving surgical treatment establishes clearly the efficacy of thoracoplasty as a collapse procedure in the treatment of pulmonary tuberculosis.

Particularly is the importance of thoracoplasty evident in the study of the fate of the slipping chronics unoperated upon. In our series, it is evident from Table II that the presence of progressive disease rendered the prognosis of these patients exceedingly grave unless major surgery was undertaken. The fate of the control group should thus remove any doubt which may exist concerning the value of surgery and its absolute necessity as a therapeutic measure in this type of patient.

The analysis of the fate of the good chronics who refused operation also throws some light upon the question of how good is the "good chronic." It is clear from Table III that a small percentage of these individuals were sufficiently fortunate to become improved or closed without major surgery. A slightly smaller group remained unchanged. Yet nearly half of these good chronics, in our experience, were destined to become either worse or dead, within a four-year period. We are aware, of course, that the experience of those observers dealing with selected groups of a better economic status and a higher level of intelligence, may be at variance with ours, yet we believe that our control group more accurately portrays the true fate of the usual good chronic. We know, in addition, that those good chronics unoperated upon, who are classified as "worse," are actually at this moment slipping chronics, and that many of those labeled as "unchanged" are destined to become slipping chronics in the near future. This not only renders the immediate prognosis of these individuals grave, but also materially reduces the possibility of recovery even should thoracoplasty eventually be performed.

It is evident, therefore, that the "good chronic" is after all only relatively good. Despite his excellent general condition at the moment, the existence of his cavity subjects him to all of its potential dangers and to a hazardous future. To delay operation in the good chronic in

the hope that he will become closed without major surgery; or to procrastinate until he has begun to slip is unwarranted and dangerous, since it is in the good chronic group that thoracoplasty produces its most brilliant results and its fewest failures.

It is evident from the preceding discussion that the fate of patients is dependent to a great extent upon the qualitative character of their disease. Table IV, which summarizes and compares the present physical status of the good and slipping chronics within the thoracoplasty and

TABLE IV

COMPARISON OF THE STATUS OF THE "GOOD CHRONICS" AND "SLIPPING CHRONICS" WITHIN THE THORACOPLASTY AND REFUSAL GROUPS

	THORACOPLASTIES				REFUSALS			
	GOOD CHRONICS 43 PATIENTS		SLIPPING CHRONICS 42 PATIENTS		GOOD CHRONICS 32 PATIENTS		SLIPPING CHRONICS 26 PATIENTS	
	%		%		%		%	
Closed	70	75	43	57	16	25	4	8
Improved	5		14		9		4	
Unchanged	5	5	7	7	22	22	4	4
Worse	2	14	12	29	28	47	42	77
Dead	12		17		19		35	
Untraced	7		7		6		12	

control groups, is intended to clarify and emphasize the importance of the qualitative classification in evaluating the prognosis of patients with and without surgery.

It has already been pointed out that the prognosis of patients refusing operation is in general unfavorable. The table shows clearly, however, that the prognosis is far more serious if the patient is a slipping chronic than if he is a good chronic. Within the four-year period, for example, 77 per cent of the slipping chronics were worse or dead, in comparison to 47 per cent of the good chronics, and the mortality rate of the slipping chronic group was twice as great as that of the good chronic group (35 per cent compared with 19 per cent). Similarly, while one out of every four good chronics (25 per cent) did well without operation, this was true of only one in twelve (8 per cent) of the slipping chronics. The failure to recognize this important qualitative classification may well explain many prognostic errors and certain of the discrepancies existing in the statistics of various authors, with respect to the fate of patients with pulmonary cavities.

Similarly, the prognosis of patients who receive thoracoplasty is dependent to a great extent upon whether or not they are good or slipping chronics. The table shows, for example, that 70 per cent of the good chronics were closed after operation in contrast to 43 per cent of the slipping chronics. Although 29 per cent of the slipping chronics were worse or dead within four years after operation, this was true of only

14 per cent of the good chronics. Finally, it will be noted that the 19 per cent mortality rate of the slipping chronic group was one-third greater than the 12 per cent mortality rate of the good chronic group. It is apparent that the utilization of a qualitative classification permits of more accurate prognosis. It explains in part certain of the variations to be found in the end-results of different surgeons. Obviously, the surgeon operating upon a group of patients consisting largely of good chronics will not only have a lower mortality rate, but also a much higher percentage of favorable results than the surgeon operating upon a large number of slipping chronics. To evaluate the results of thoracoplasty solely on the basis of undifferentiated statistics may produce an erroneous picture of its true efficacy as a therapeutic procedure.

FUNCTIONAL STATUS

The functional status of the patients in this series has been considered separately, in order to avoid a certain degree of confusion which might arise particularly with respect to the control group. It is well known, of course, that there is no necessary correlation between the extent of a patient's pulmonary disease and his functional capacity. An individual with progressive disease who is "curing" in the sanatorium may yet be sufficiently asymptomatic and in good enough condition to be able to work. Merely to classify this patient as "able to work," obviously imparts an inaccurate picture of the patient's functional status at the moment. The fact that he is in the sanatorium renders him functionally inactive. We have, therefore, adopted the following classification which accurately presents the functional status of our patients at the time of the last observation.

Able to Work.—This group includes those patients who are closed or improved (and unchanged in the case of the refusal group) and who, in the opinion of the examiner, are able to work full or part time (the latter at least three to four hours daily). No patient is included who is at present under treatment, either in the sanatorium or at home, regardless of his general condition. In each table is also indicated the number of patients actually at work. Because of present economic circumstances, the number of patients working is an unreliable guide in estimating functional capacity.

Well But Unable to Work.—In this group are included those patients who are closed or improved, but who are unable to work, usually because of diminished vital capacity, weakness, easy fatigue, or because the extent of their original disease renders it inadvisable for them to consider active employment. These are the patients who are functionally incapacitated even though satisfactory anatomic healing of their disease has occurred.

Curing.—This group includes all patients still "curing," either at home or in the sanatorium; and all patients previously classified as worse, regardless of their general condition. These patients require treatment and are, therefore, for all practical purposes functionally inactive. The group thus comprises those individuals unable to work because of the progression of their disease, or because they are still undergoing treatment.

TABLE V

PRESENT FUNCTIONAL STATUS OF THE COMPLETE THORACOPLASTY AND REFUSAL GROUPS

	THORACOPLASTIES 85 PATIENTS		REFUSALS 58 PATIENTS	
	NO.	%	NO.	%
Able to work	40[1]	47	9[2]	16
Well but unable to work	8	9	1	2
Curing	19	22	28	48

[1]Sixteen patients (19 per cent) working full or part time.
[2]Three patients (5 per cent) working full or part time.

The present functional status of the thoracoplasty group and its control group is presented in Tables V and VI.* Since the generalizations to be drawn from the study of these groups are in many respects similar to those already noted in the consideration of the physical status of these patients, the tables will require no extended discussion.

Results in General (Table V).—Forty-seven per cent of the thoracoplasty group are at the present time able to resume economic activity, in contrast to 16 per cent of the control group.† Forty-eight per cent of the patients who refused operation are still under treatment at home or in the sanatorium, while this is true of only 22 per cent of the thoracoplasty group. Nine per cent of the thoracoplasty group are at the present time well, but unable to work for the reasons enumerated above, nor can they, in our opinion, expect to resume economic activity. Yet thoracoplasty in these patients has been of value, since it has permitted them to leave the sanatorium and to resume, in full or in part, and without danger to others, their usual social existence. Few of these patients have expressed dissatisfaction with this end-result.

It is apparent, therefore, that not all of the patients operated upon have been restored to functional activity. In our experience this occurred in slightly less than half the cases. Yet by comparison with the fate of the control group, thoracoplasty was distinctly worth while, since it increased by more than four times the number of patients restored to economic usefulness, and decreased by more than half the number of

*The patients dead or untraced are omitted from these tables. Their number may be secured from Tables I-III.

†Included in the occupations engaged in by the thoracoplasty patients who are working are stenographer, salesman, musician, insurance agent, public health nurse, gasoline station attendant, hospital orderly, store clerk.

patients under treatment at home or occupying sanatorium beds, because of a persistently positive sputum or an open cavity.

Results in the Slipping Chronic Group (Table VI).—Thirty-one per cent of the patients operated upon are able to work at the present time. An equal number, 31 per cent, are still under treatment and an additional 14 per cent, although closed or improved, cannot hope to resume functional activity. To judge the results of thoracoplasty solely from these figures, however, would be misleading, since the table shows that of the slipping chronics who refused operation only 4 per cent (1 patient) are able to work, while 50 per cent of the group are still "curing" at home or in the sanatorium. In the slipping chronic group, therefore, thoracoplasty increased by eight times the number of patients restored to functional usefulness, and decreased by one-third the number of patients still under treatment.

TABLE VI

PRESENT FUNCTIONAL STATUS OF THE "GOOD CHRONICS" AND "SLIPPING CHRONICS"

	THORACOPLASTIES				REFUSALS			
	GOOD CHRONICS 43 PATIENTS		SLIPPING CHRONICS 42 PATIENTS		GOOD CHRONICS 32 PATIENTS		SLIPPING CHRONICS 26 PATIENTS	
	NO.	%	NO.	%	NO.	%	NO.	%
Able to work	27[1]	63	13[2]	31	8[3]	25	1[4]	4
Well but unable to work	2	5	6	14	1	3	0	0
Curing	6	14	13	31	15	47	13	50

[1]Eleven patients (26 per cent) working full or part time.
[2]Five patients (12 per cent) working full or part time.
[3]Two patients (6 per cent) working full or part time.
[4]One patient (4 per cent) working part time.

Results in the Good Chronic Group (Table VI).—It is in the good chronic group that thoracoplasty produced the most satisfactory functional results. Sixty-three per cent of the patients operated upon are, at the present time, able to work, in contrast to 25 per cent of the control group. Although 14 per cent of the group operated upon are still under treatment, this is true of 47 per cent of the individuals who refused operation. In the good chronics, therefore, thoracoplasty increased by two and one-half times the possibility of restoration of functional capacity, and decreased by more than three and one-half times the number of patients still "curing." The large number of good chronics who are under treatment, either at home or in the sanatorium, should be noted; the percentage is not far different from that of the slipping chronic group (50 against 47 per cent). This emphasizes again the unsatisfactory course of the unoperated good chronic and the value of thoracoplasty as a therapeutic measure.

It is apparent, therefore, that in comparison with the fate of the control group, thoracoplasty is an effective therapeutic procedure in restor-

ing patients to economic activity; that the most satisfacory results are found in the good chronic group; that thoracoplasty is of limited value in restoring the slipping chronics to functional activity, yet this is a distinct improvement over the fate of the patients not operated upon; that in both the good and slipping chronic groups thoracoplasty is of importance in reducing the number of patients requiring prolonged treatment of their disease, either at home or in the sanatorium; and finally, that the statistics of the end-results of thoracoplasty with respect to functional status depend to a great extent upon the proportion of good chronics and slipping chronics in the group.

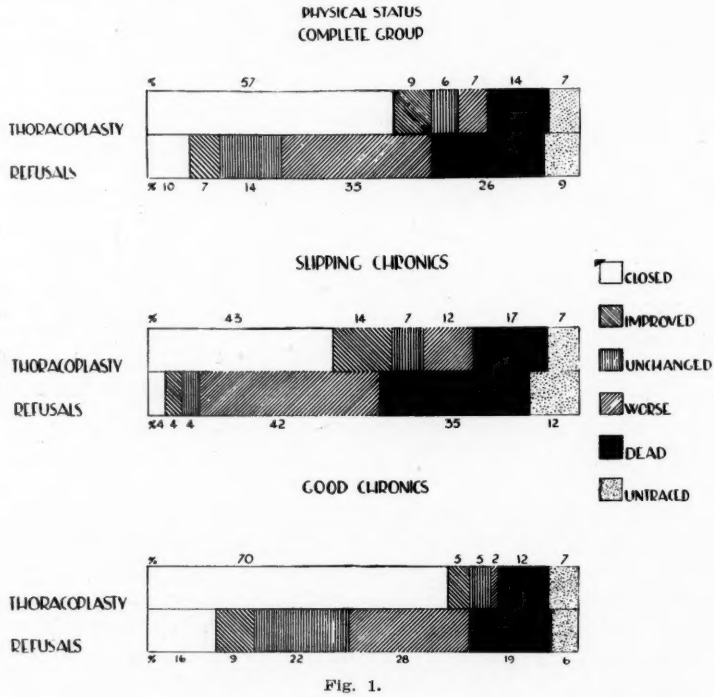

Fig. 1.

EFFECT OF DELAYING OPERATION

Ten patients in the group under consideration accepted thoracoplasty one to three years after the initial recommendation. These patients have all been operated upon during 1935 and 1936, and though the period of time which has elapsed is too short to permit any evaluation of the final result, certain points with respect to their condition at the time of operation are of interest.

Of these patients, two were slipping chronics and eight were good chronics at the time operation was recommended. Both of the slipping

chronics at the time of operation had definite extension of their disease and an increase in constitutional symptoms. Both were unquestionably poorer risks when operated upon than they were one to three years earlier.

Of the eight good chronics, four were in essentially the same condition when operated upon as at the time of the original thoracoplasty recommendation. In the remaining four instances, however, the patients, instead of being good chronics, were slipping chronics when surgery was finally accepted. In these four patients there was not only extension

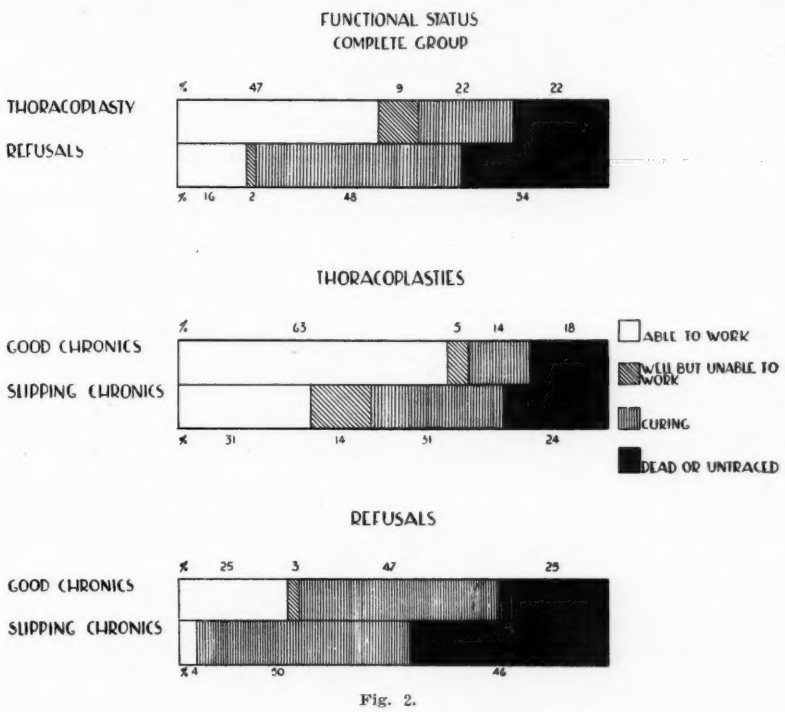

Fig. 2.

of the disease, but a lowered vital capacity and a recurrence of constitutional symptoms. In two of the patients it was necessary to perform complete thoracoplasty where partial thoracoplasty would previously have sufficed.

Certainly no gain accrued to these ten patients by delaying operation. Not only did they spend many additional years under active treatment, but in half of the cases the risk of operation was increased and the possibility of recovery was definitely lessened. These ten patients emphasize again the point previously made, that procrastination in instituting treatment is unwarranted and dangerous.

SUMMARY

1. This study compares the results of thoracoplasty performed upon 85 patients during the years 1932 to 1934, inclusive, with the fate of a control group of 58 patients who refused operation during the same period. The results are summarized in graphic form in Figs. 1 and 2.

2. For more accurate comparison both the thoracoplasty group and the control group are differentiated into "good chronics" and "slipping chronics." The importance of such a qualitative classification in the analysis of thoracoplasty statistics is pointed out.

3. The analysis shows that without thoracoplasty the course of the disease in the good chronics is very different from that in the slipping chronics, but that thoracoplasty notably improves the prognosis of each group, both in regard to the healing of disease and in the restoration of work capacity.

4. It is emphasized that to delay operation in the good chronic in the hope of spontaneous recovery is unjustifiable.

5. A separate study of 10 patients whose operation was delayed from one to three years after it was first advised, contributes further evidence that procrastination is hazardous.

We are indebted to Mrs. Carrie E. Lewis, of the Bureau of Tuberculosis of the Cleveland Health Department, and to Dr. Joseph Stocklen, of the Sunny Acres Sanatorium staff, for valuable aid in the follow-up studies.

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PARAFFIN PACK AND THORACOPLASTY IN THE CLOSURE OF LARGE APICAL CAVITIES*

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THE closure of large apical cavities remains one of the major problems of the surgery of pulmonary tuberculosis. Their tendency to retreat against the spine or mediastinum and remain patent following thoracoplasty is too well known to require description, as are also the many extensions of the standard thoracoplasty which have been devised to collapse them. These include the resection of the full lengths of the upper ribs and cartilages (Alexander), as well as of the transverse processes, and the exertion of additional direct pressure by gauze packs (Sauerbauch, Casper), subcostal balloons (Harvey) and postthoracoplasty paraffin packs. As early as 1914, Wilms performed an apicolysis at the same time as the thoracoplasty and packed in the segments of resected ribs.

During the past several years I have been treating such lesions by an initial paraffin pack, and, if this failed to render the sputum negative, by removal of the pack and the substitution of a secondary thoracoplasty. I was led to adopt this sequence of operations by the observation that the thoracoplasties performed after the removal of paraffin packs gave a high percentage of favorable results, and by the following theoretical considerations: (1) The initial paraffin pack is a less dangerous procedure than thoracoplasty. (2) It has a fair chance of closing the cavity and rendering the sputum negative. (3) If it does not, it so decreases the size of the cavity and so stabilizes the mediastinum that the secondary thoracoplasty is much more likely to be effective. (4) Thoracoplasty, secondary to paraffin pack, is far safer than primary thoracoplasty, because the decrease in the amount of sputum lessens the chances of spill, and the thickening of the mediastinum prevents paradoxic respiration.

At the present time, I am able to report 16 cases with large apical cavities in which this plan of treatment has been carried to completion.

The results in this group are as follows:

	CASE	PER CENT
Sputum rendered negative by paraffin pack	6	37.5
Sputum rendered negative by secondary thoracoplasty	7	43.75
Improved and clinically well. Sputum still positive after secondary thoracoplasty	2	12.5
Worse—secondary thoracoplasty impossible on account of spread to opposite lung	1	6.25

*Read before the Nineteenth Annual Meeting of the American Association for Thoracic Surgery at Rochester, Minnesota, May 4, 5, 6, 1936.

Combined results in 16 cases with large apical cavities:

	CASE	PER CENT
Sputum rendered negative	13	81.25
Improved	2	12.5
Worse	1	6.25
Dead	0	0

PROCEDURE

Before a large cavity is treated by direct surgical compression, everything possible should be done to permit it to decrease in size. Absolute bed rest, artificial pneumothorax, even though only partially successful, and phrenic nerve block should be used and persisted in as long as improvement is progressive. By these measures an extremely difficult surgical problem can frequently be rendered simple and the chances of ultimate cure greatly increased. In these cases, if the lower lung is free of disease, temporary diaphragmatic paralysis is preferable to permanent. As soon as the cavity has ceased to grow smaller, surgery should be resorted to, for the dangers of hemorrhages and spill, and the development of extrapulmonary complications are always present.

In all save two of the cases in this series, the paraffin pack was inserted from the rear. It is the preferable approach. In the presence of large thin-walled cavities, stripping must be done with great care to avoid tearing the lung. In many a very imperfect collapse was obtained, a circumstance which had little bearing on the result following thoracoplasty. In the more recent cases, as the technique of stripping improved, complete inversion packs were frequently obtained. It is my impression that the results in these have been less satisfactory than when the lung was merely pressed against the mediastinum, or at least left adherent at the extreme apex. In the former, the cavity, while markedly decreased in size, was shoved down into the thorax where the compression from thoracoplasty was less effective.

The pack should be left in place for at least three months, and as long thereafter as there seems to be a possibility that the sputum will become negative.

The secondary thoracoplasty should be partial or complete, depending upon the distribution of the lesions in the lung. In this series, six of the ten thoracoplasties were complete; in all save one, the full length of the upper three or four ribs and portions of the cartilages were removed. The upper stage of the thoracoplasty was done first and the pack removed before resection of the upper two ribs. This provides better exposure for their removal and prevents undue pressure on the trachea and vessels during the operation.

COMPLICATIONS

The chief theoretical objection which can be raised against this sequence of operations is that the complications incident to the pack

might be more important than the advantages to be gained from it. In this series, that has not proved to be the case. While complications have occurred, in only one instance, that in which disease developed in the opposite lung several months after the extrapleural operation, have they prejudiced the final results. The danger of postoperative spill or atelectasis following the pack is definite, but rarely serious, and is not greater in such cases than in those following primary thoracoplasty. It is largely offset by the greater safety of the secondary thoracoplasty, performed at a time when the decrease in the amount of sputum and the stabilization of the mediastinum have practically eliminated these important dangers. In this series the complications were as follows:

Postoperative atelectasis of homolateral lower lobe following pack—1 case—patient cured by complete thoracoplasty performed three months later.

Postoperative pneumonic exacerbation of disease already present in homolateral lower lobe—1 case—patient cured by thoracoplasty performed three months later.

Spread of disease into opposite lung three months after insertion of pack—1 case.

Perforation of pack and expectoration of paraffin—3 cases.

The cases of perforation of the pack demand special consideration. In my opinion it is not a serious complication. In one of the cases, the pack has been removed and thoracoplasty performed. The bed of the pack, even though a definite fistula was present, was closed without drainage. While the sputum is still positive, there is no evidence that the complication has affected the result. In the other two cases, the patients, after having had a negative sputum for two years, have recently begun to expectorate small pieces of paraffin. They have no other symptoms, no fever, and their tuberculosis is still apparently arrested. If they continue to raise paraffin, the pack will be removed. I have not yet decided whether I shall perform a thoracoplasty at the same time, or merely remove the paraffin and make a primary closure of the bed.

RESULTS IN OTHER CASES TREATED BY PARAFFIN PACK

In conjunction with the statistics already presented, it seems advisable to include those on the remaining cases which have been treated by paraffin pack. The first group has included those with large apical cavities in which the pack was used in preparation for thoracoplasty. The remaining ones fall naturally into two groups: those with lesser lesions in which thoracoplasty would have been possible, and those in which disease in the opposite lung, or low vital capacity, contraindicated the major operation.

There were 15 cases in which packs were used to collapse smaller fibrous apical lesions and cavities, cases in which thoracoplasty was feasible and its success probable. The results in these cases are as follows:

	CASES	PER CENT
Sputum rendered negative by pack	9	60.0
Sputum rendered negative by secondary thoracoplasty	2	13.22
Improved—preexisting disease in opposite lung	1	6.66
Worse—preexisting disease in opposite lung and T. B. enteritis	1	6.66
Dead—preexistent tuberculosis enteritis Thoracoplasty not performed	1	6.66
Dead—nontuberculous lobar pneumonia	1	6.66

Combined results in 15 cases with smaller apical cavities:

	CASES	PER CENT
Sputum rendered negative	11	73.32
Improved	1	6.66
Worse	1	6.66
Dead	2	13.32

A combination of these two groups in which the paraffin pack was used, either as a preliminary operation or in preference to thoracoplasty, gives the following results:

	CASES	PER CENT
Sputum rendered negative by pack	15	48.4
Sputum rendered negative by secondary thoracoplasty	9	29.0
Improved	3	9.7
Worse	2	6.45
Dead	2	6.45
COMBINED RESULTS		
Sputum negative	24	77.4
Improved	3	9.7
Worse	2	6.45
Dead	2	6.45

The third group is composed of thirteen miscellaneous cases in which the pack was used because thoracoplasty was definitely contraindicated. In two, it was used to supplement thoracoplasty. In one, the sputum was rendered negative. In two cases bilateral apical packs were inserted in an attempt to close large, fibroid, apical cavities. In each, one pack was inserted from in front and one from behind. In each, because the lung was injured at the time of operation, the anterior pack extruded and was removed and a local thoracoplasty performed. Both patients were improved, but in neither was the sputum rendered negative. In one case the pack was used in preference to thoracoplasty because the patient had definite hemophilia. The pack perforated and was eventually removed. He has improved and is now ambulatory. The opposite lung is still normal, but his ability to withstand a thoracoplasty is very questionable. It will be attempted. In two cases the pack was used because extensive healed tuberculosis in all lung fields had so reduced the vital capacity that there was dyspnea at rest, and thoracoplasty was conse-

quently out of the question. In one of these the pack perforated, its bed became badly infected, and the patient died. The remaining six cases had extensive active disease in the opposite lung. Spread of the disease rendered two of these worse and the others were improved, but not cured by the operation.

SUMMARY AND CONCLUSIONS

1. The failure of thoracoplasty to close large apical cavities accounts for many of the partial successes following the operation.

2. The results in this group of cases can be markedly improved by using an initial paraffin pack.

3. In a certain number of cases, this will close the cavity and render the sputum negative.

4. In those in which it fails to do so, it so decreases the size of the cavity and the amount of sputum, and so stabilizes the mediastinum that a secondary thoracoplasty is far safer and far more likely to be effective.

5. The sputum was rendered negative in twelve of sixteen cases (75 per cent) of large apical cavities, treated by the sequence of operations.

6. In fifteen cases of smaller lesions in which the pack was used in preference to thoracoplasty, the sputum was rendered negative in twelve, or 80 per cent. Two of these are still to have thoracoplasty.

7. Of the thirty-one cases in which thoracoplasty was not contraindicated and the pack used either in preparation for, or in preference to it, the sputum was rendered negative in twenty-four, or 77.4 per cent.

8. In a group of thirteen miscellaneous cases in which the pack was used because thoracoplasty was definitely contraindicated because of bilateral cavities or extremely low vital capacity, most have been improved but none cured.

PARTIAL RESECTION OF THE LOWER SCAPULA AS AN AID IN COMPRESSING APICAL TUBERCULOUS ABSCESSES AND IN CONSERVING VITAL CAPACITY*

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IN THE cure of pulmonary tuberculosis, the paravertebral apical abscess presents one of our most difficult problems. It may be stated categorically, that unless it can be obliterated by compression or by re-

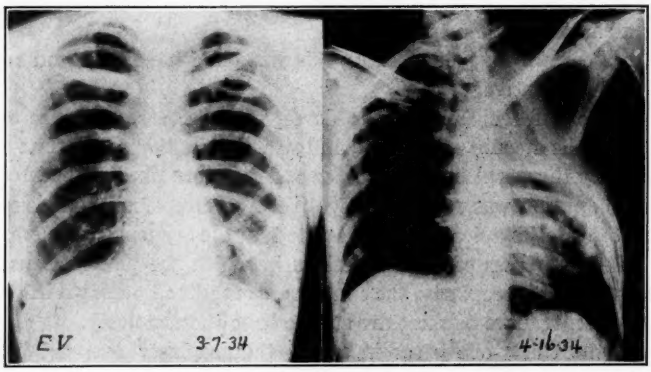

Fig. 1.—Patient E. V. Bilateral pulmonary tuberculosis with large left apical abscess. Resection of upper five ribs and lower fourth of left scapula in a one-stage operation, April 3, 1934, produced complete obliteration of cavity. Patient remains well.

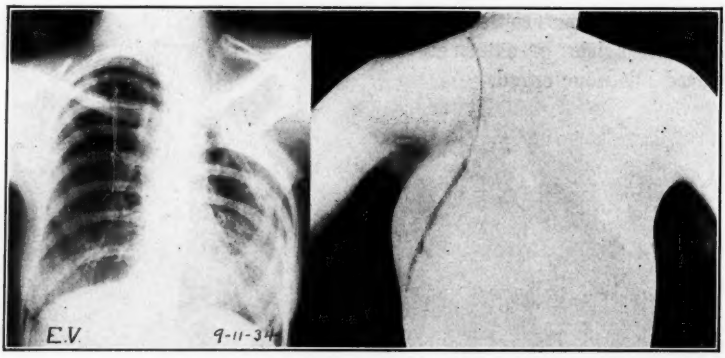

Fig. 2.—Patient E. V. (a) Appearance of chest five months later. (b) Deformity is not conspicuous, although probably slightly more pronounced than following the usual thoracoplasty.

expansion of the surrounding normal pulmonary tissue, it will remain as a focus of disease which nullifies all efforts to effect a cure. The

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reasons for this are obvious. Destruction of the apical pulmonary tissue has left a "dead space," the walls of which are formed laterally and above by the rigid thoracic cage, and below by a thick fibrous floor, which is frequently moored securely to the ribs by dense fibrous tissue. Obliteration of this "dead space" is essential to complete healing.

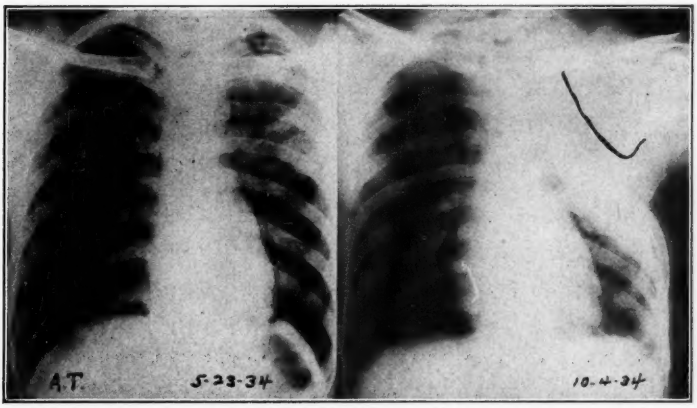

Fig. 3.—Patient A. T. (a) Unilateral pulmonary tuberculosis with multiple small abscesses, limited largely to left apex. (b) Resection of upper five ribs and lower half of scapula (Fig. 9c) in one-stage operation on September 18, 1934, with conservation of lower uninvolved lung. Patient is sputum-free.

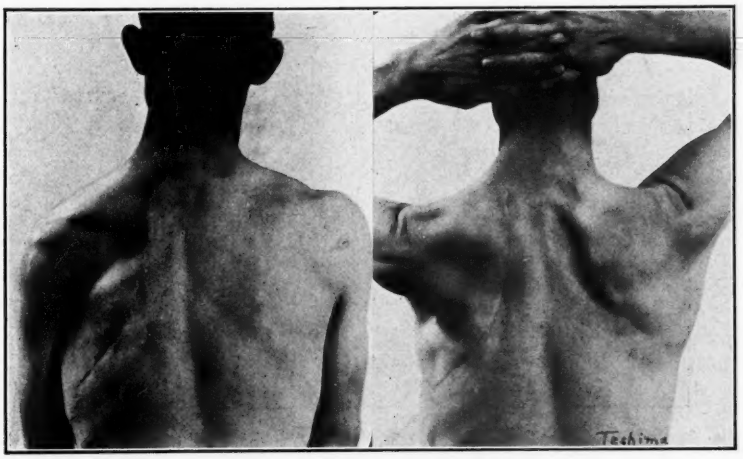

Fig. 4.—Patient A. T. Appearance of patient following partial resection of upper five ribs and of lower half of scapula. Motion of arm and shoulder only slightly impaired.

Not infrequently such an abscess, though constituting the most intractable focus, is only part of a more generalized process involving ipsilateral and contralateral lobes. Under such circumstances, it is imperative to conserve as much of the uninvolved lung and of an already greatly reduced vital capacity as possible. An old-fashioned Sauerbruch

procedure, such as a complete unilateral thoracoplasty, may be definitely contraindicated because of bilateral disease. Obviously, the most logical procedure is some form of partial thoracoplasty. Resection in their entirety of the upper three ribs from vertebral to sternal attachments, the partial resection of the fourth, fifth and, possibly, sixth ribs, and the re-

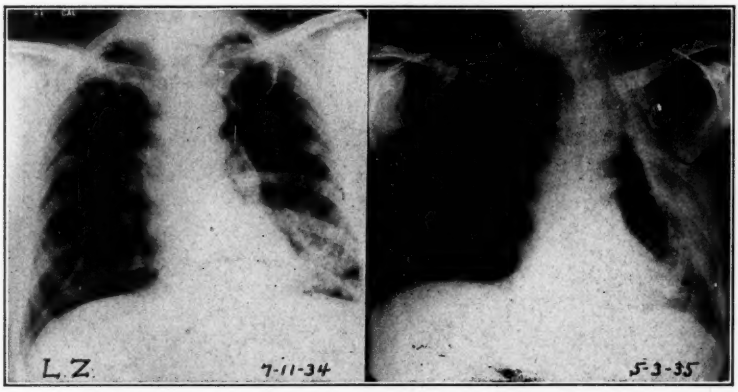

Fig. 5.—Patient L. Z. Large apical abscess on left, obliterated by complete resection of first three ribs at first-stage operation on February 16, 1935, and by resection of next three ribs and one-half of scapula at second-stage operation on March 7, 1935. Patient is free of tubercle bacilli and is well.

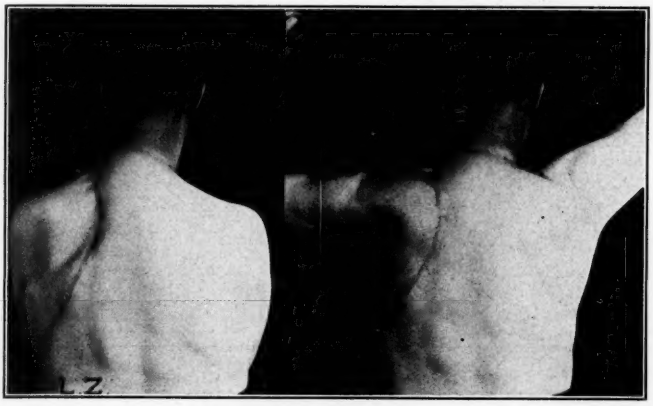

Fig. 6.—Patient L. Z. Only moderate deformity, and moderate limitation of motion.

section of the transverse processes of the third, fourth and fifth vertebrae, may be necessary to effect complete obliteration of the cavity.

The greatest obstacle to the maximum benefit from such a partial thoracoplasty has been the scapula. If only five ribs are resected, the scapula effectively prevents the complete falling in of the posterior thoracic wall. If only six ribs are removed, the lower angle of the

scapula rides on the seventh rib, causing localized pain and elevation of the shoulder. The resection of seven ribs may be a too formidable pro-

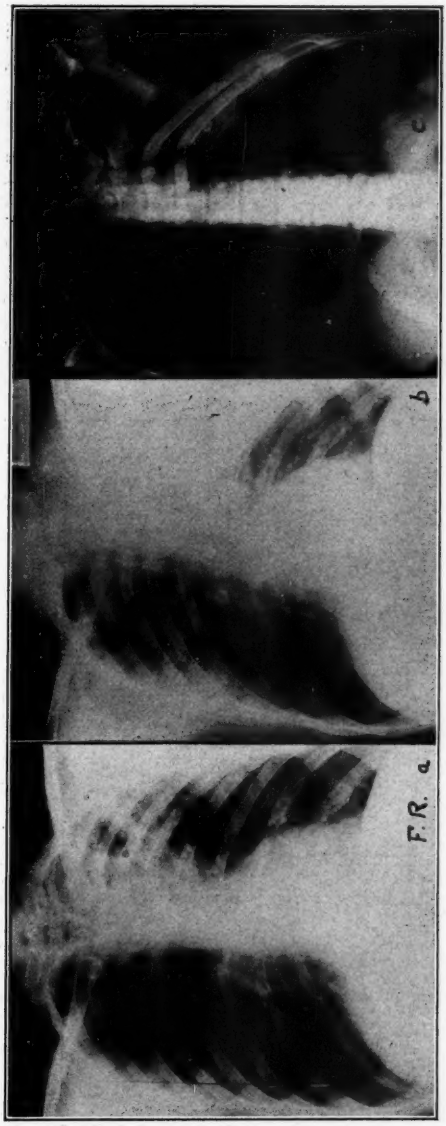

Fig. 7.—Patient F. R. Large apical abscess obliterated by resection of upper four ribs and lower half of scapula in one stage, with conservation of lower uninvolved lung.

cedure for patients with the type of general involvement under discussion, and the additional sacrifice of good lung and of vital capacity is unwarranted when the sole object is obliteration of an apical cavity.

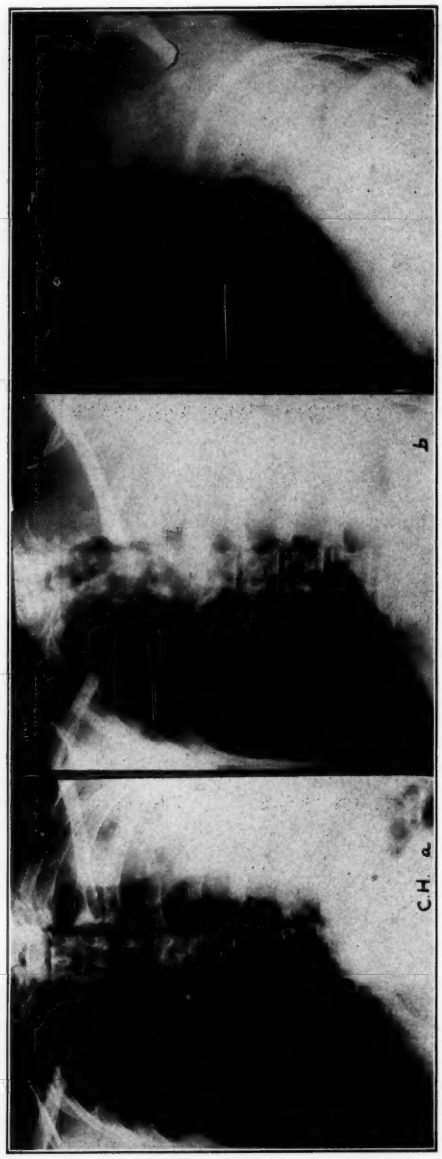

Fig. 8.—Patient C. H. Destruction with cavitation of most of left lung by tuberculosis. Trachea and mediastinum lie well to left of midline. Heart occupies lower portion of left thorax. Upper lung compressed by partial resection of upper four ribs at first operation (Fig. 8b), and of fifth and sixth ribs and one-half of lower scapula (Fig. 9b) in second operation.

Such reasoning led to the resection of the lower third of the scapula as an alternative procedure to resection of the sixth and seventh ribs. The first, second, and third ribs are removed completely, together with the posterior third of the fourth and fifth ribs. The resection of the lower one-third to one-half of the scapula is easily performed, with a minimum of bleeding, by an incision around the angle, elevation of the periosteum and attached muscles by a broad, sharp elevator, and excision of the denuded bone by rongeurs. Enough bone is removed to permit the scapula to fall easily into the space provided by resection of the upper five ribs.

This operation for the collapse of an apical cavity may be done in one or two stages. Following the complete resection of the upper three ribs

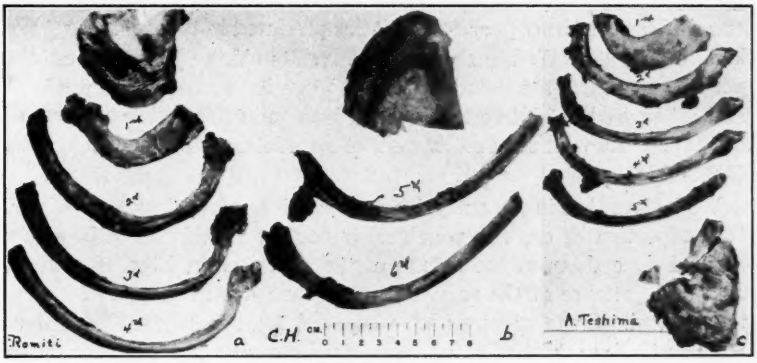

Fig. 9.—Illustrating lengths of ribs and amount of scapula resected in three patients, F. R.; C. H.; and A. T. Compare with Figs. 3, 4, 7, and 8.

and portions of the fourth and fifth, the menace of paradoxical respiration is always great, depending upon the thickness and rigidity of the parietal pleura. Accordingly, if removal of the upper three ribs causes active paradoxical movement of the mobilized chest wall, the operation is terminated, and at the second stage, ten to fourteen days later, the fourth and fifth ribs together with the lower portion of the scapula are removed. If the paradoxical movements are only moderate, due to fibrous rigidity of the visceral and parietal pleurae, the operation may be completed in one stage. In fact, the falling in of the small scapula may effectually prevent paradoxical respiration and may, therefore, permit completion of the operation in one stage. Further experience with the operation will be necessary to determine this point.

CLOSED AND OPEN INTRAPLEURAL PNEUMONOLYSIS* RESULTS IN 111 AND 29 CASES, RESPECTIVELY

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THE surgical division of pleural adhesions in cases of inadequate therapeutic pneumothorax for pulmonary tuberculosis has been the subject of many articles, some of which have evaluated the operation in terms of almost unqualified praise. Few articles have damned the operation, but a considerable number of internists are opposed to it because of the relatively poor results it has produced in their patients. We believe that a detailed analysis of the results obtained in our first 111 patients for whom the closed method of operation was used between 1927 and 1934 may be of interest. We shall include in this article a report of our experience with our first 29 cases of open intrapleural pneumonolysis (1930-1934).

We believe that in the operation of closed intrapleural pneumonolysis, the proportion of cases in which all important adhesions can be safely divided depends upon the nature of the adhesions and the experience, skill and patience of the surgeon, rather than upon the type of instruments used. Some surgeons have changed from instrument to instrument in the hope that what is necessarily frequently a difficult operation may be made easy. We prefer the two-piece, galvanocautery instrument of the Jacobaeus-Unverricht type and have used it in all but a few of our patients; in these, a two-piece electrosurgical instrument was used.

Many surgeons avoid undertaking to divide broad or complex adhesions while they are acquiring their early experience with the operation, and then gradually become more bold. We failed to divide any adhesion in 15.4 per cent of our early cases, divided some but not all of the adhesions in 57.7 per cent, and divided all the adhesions in 26.9 per cent. In our later cases, which included many we would not have undertaken to operate upon during our early experience, the corresponding percentages were 23.5, 36.5, and 40.0.

We have divided all 111 cases into groups in which operation was performed when the pneumothorax had existed for six months or less, from six to twelve months, and for additional six-month periods. The percentage of complete surgical division of adhesions increased (with the exception of one group) as the age of the pneumothorax increased. A probable explanation is that the indication for early operation is often

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sufficiently urgent to justify an attempt to divide adhesions that are from the roentgenologic point of view only questionably suitable for the operation. It was also found, as might be expected, that the percentage of complicated adhesions was considerably higher in the young than in the old pneumothorax cases. We found, after inspection of the adhesions with a thoracoscope, that 23.5 per cent of those in pneumothoraces, six months or less in age, were not at all suitable for division; for groups of successively older pneumothoraces, the percentages are 19.5, 33.3, and 0.0. The operation was performed in the 111 cases an average of 8.6 months after the induction of pneumothorax.

In our experience relatively few pneumothorax patients have adhesions that are suitable for closed or open intrapleural pneumonolysis. During a five-year period at the Michigan State Sanatorium, these operations were used in only 14.8 per cent of the 627 cases (i.e., 542 patients, of whom 85 had bilateral pneumothorax) in which pneumothorax was undertaken. The percentage would, of course, be somewhat higher for the group of patients with only a partial pneumothorax. We believe that in some clinics adhesions are divided that are not adversely affecting pulmonary collapse, or that would not do so if permitted to stretch, as so many adhesions do with the increasing duration of the pneumothorax.

Among the 111 patients the adhesions were completely divided in 37.0 per cent, were incompletely divided in 41.4 per cent, and were found at operation to be wholly unsuitable for division in 21.6 per cent. At attempt had been made before operation to estimate from a study of the roentgenograms the probable success of operation. The chance of complete success was considered good in two of the 24 cases in which the adhesions were actually found unsuitable for division (in one of the two cases, however, because of apparently active pleural tubercles), fair in seven (the adhesions in three of these were not divided because of pleural tubercles), questionable in 14, and poor in one. The preoperative prediction in the 46 cases in which the adhesions were incompletely divided was good in 11, fair in 14, questionable in 18, and poor in 3. The preoperative prediction in the 41 cases in which adhesions were completely divided was good in 25, fair in 10, questionable in 6, and poor in none.

Although the indications and contraindications to closed intrapleural pneumonolysis will not be considered in detail in this article, brief mention will be made of the choice between this operation and phrenic paralysis or thoracoplasty. If a partial pneumothorax seems unlikely to succeed in effecting adequate collapse of the pulmonary lesions, a phrenic paralysis should be used in preference to intrapleural pneumonolysis, if the cavity is suitable for the former operation. In 38 (34.2 per cent) of our 111 patients, a temporary or permanent phrenic paralysis had been produced an average of 7.2 months before the pneumonolysis op-

eration was performed. In the following types of cases, however, consideration should be given to the choice of intrapleural pneumonolysis in preference to phrenic paralysis: (1) When an adhesion that is apparently suitable for division immediately overlies a soft, actively progressing lesion, especially one that includes a superficial cavity, the tension exerted upon the lesion through the adhesion during every respiration is potentially dangerous, even if the diaphragm is paralyzed. (2) When the adhesion exerts a horizontal pull upon the pulmonary lesions, paralysis and elevation of the diaphragm are not likely to be as effective as division of the adhesion. (3) When active lesions in the contralateral lung may require a phrenic paralysis, this operation should not be used in preference to intrapleural pneumonolysis for the lesions of the pneumothorax lung, unless the physician is willing to risk a bilateral phrenic paralysis in the presence of a unilateral pneumothorax.

Whether a patient having an inadequate pneumothorax that presents adhesions suitable for the closed type of intrapleural pneumonolysis should have this operation, or a thoracoplasty (assuming that no other operation is suitable to close the cavity), has been much debated. A number of surgeons feel that pneumonolysis is too complicated and uncertain in its effect for adoption and that, therefore, thoracoplasty is the better operation. In support of this view is the fact that pneumonolysis is not an end in itself. It aims only to create an adequate pneumothorax. The patient is subjected to the combined risk of pneumonolysis and the subsequent pneumothorax, which must be weighed against the risk of thoracoplasty. Most surgeons, however, who are personally familiar with pneumonolysis, prefer this operation to thoracoplasty, provided the particular adhesions indicate that they probably can be divided without exceptional difficulty, and that the pulmonary lesions are suitable for complete control by the adequate pneumothorax that a successful pneumonolysis produces. But a patient whose pneumonolysis operation is likely to be unusually difficult and dangerous, or whose lesions do not offer a good prospect for an uncomplicated course and successful termination of pneumothorax treatment, should have a thoracoplasty, if not contraindicated, in preference to pneumonolysis. In many cases for which pneumonolysis is used, thoracoplasty is, however, definitely contraindicated, chiefly because of progressive lesions in the contralateral lung. Thirty-one of our 111 patients had open cavities in the contralateral lung at the time of the pneumonolysis operation.

Our patients have suffered no operative complications, and few postoperative complications of importance. In no case was there an operative or postoperative hemorrhage. A varying degree of temporary emphysema of the thoracic wall was a constant postoperative finding. In one case in which the adhesions had been completely divided, air continued to escape from the pneumothorax as fast as it was introduced,

and, in spite of careful fluoroscopic control, the pneumothorax was lost. Six other patients had considerable thoracic wall emphysema, but the pneumothorax was maintained and temporary discomfort was the only troublesome symptom.

Transient serous effusion occurred in 31, or 27.9 per cent, of the cases. In six of these no attempt had been made to divide adhesions; in 12, the adhesions had been divided incompletely; and in 13, completely. A persistent serous effusion occurred in an additional 11 patients (9.9 per cent), in six of whom it had existed before operation; in four of these 11 patients no attempt had been made to divide adhesions, in three the adhesions had been divided incompletely, and in four, completely. There had been a preoperative serous effusion in 13, or 11.7 per cent, of the 111 patients; seven of these 13 effusions disappeared soon after operation and six persisted, two becoming purulent; in two of these six patients no attempt had been made to divide adhesions, in two the adhesions had been divided incompletely, and in two, completely.

Four of our patients developed a pure tuberculous empyema after operation; two of these four empyemas developed from a preoperative serous effusion, and one occurred in a patient who had tubercles on the pleura at the time of the division of the adhesions. Tubercles were found on the pleura in at least seven of the 111 patients; in only two of these seven did the surgeon consider the indication for the division of the adhesions sufficiently vital to justify him to divide the adhesions and expose the patients to a considerably increased chance of the development of a complicating empyema; one of the two patients developed a tuberculous empyema, as just noted, and the other had no postoperative exudate. Among the five patients with pleural tubercles, whose pleura was merely inspected with a thoracoscope, but in whom no attempt was made to divide the adhesions because of the danger of tuberculous empyema, two had a serous exudate at the time of operation; the exudate disappeared promptly in both cases; three of the five patients had no exudate at the time of operation, but a serous exudate formed in two patients, persisting in one.

Mixed tuberculous and pyogenic empyema occurred in two (1.8 per cent) of our patients; both were operated upon early in our experience with intrapleural pneumonolysis. In one of these patients, a short adhesion was incompletely divided, and, in retrospect, we believe that the pulmonary cortex was probably directly damaged by the cautery; after operation a pure tuberculous empyema (from which no pyogenic organisms could be cultured for several weeks) developed, followed by a bronchopleural fistula and a mixed empyema; the patient died. The second patient's adhesions were completely divided over a huge cortical cavity; a pure tuberculous empyema developed, and a gomenol-oleothorax was instituted; eventually, the oil seeped through a needle track which became a pleurocutaneous sinus and which, in turn, infected the

tuberculous empyema with pyogenic organisms; the empyema was drained, extrapleural and Schede thoracoplasties were later performed, and the patient is now entirely well.

In addition to the case already mentioned in which a pneumothorax was lost through thoracic wall emphysema, effective pulmonary collapse was lost in four (3.6 per cent) of our patients through the gradual development of obliterative pleuritis. In three of these four patients, the adhesive pleuritis complicated a serous pleural effusion, and in one, a pure tuberculous empyema.

The results of intrapleural pneumonolysis should be considered in three categories: (1) the technical success in the division of the adhesions; (2) the effect of the operation upon the lung of the side operated upon; (3) the effect of the operation upon the patient. In any consideration of the indications for, and the results of, intrapleural pneumonolysis, it is important that the fact be constantly kept in mind that the operation is not an end in itself, but that its sole object is to produce a satisfactory pneumothorax. Complete technical success in the division of adhesions does not, therefore, assure the patient's recovery. Even a complete pneumothorax may fail to close a stiff-walled cavity, and the patient is exposed to the possibility of complications arising from both the pneumonolysis operation and the continuation of the pneumothorax to its termination. Many of the published reports of "clinical success" refer merely to the clinical effect of the first few months of pneumothorax that follow operation, and not to the later results to which such definite criteria as those of the National Tuberculosis Association are applied.

We divided all the adhesions attached to the diseased portion of the lung in 37 per cent of our 111 cases, divided some, but not all, the adhesions in 41.4 per cent, and found that the adhesions were wholly unsuited for division in 21.6 per cent. It should be noted that incomplete division of adhesions may permit a sufficient pulmonary collapse by pneumothorax to heal the lesions.

The effect of operation upon the lung and pleura of the operated side depends not only upon the degree of the technical success of the operation, but also upon any complications that may occur, and the adequacy of the postoperative pneumothorax. The general effect on the operated side was excellent in 58.5 per cent of our 58 patients whose cavities became closed, or whose noncavernous lesions were adequately collapsed by operation. The effect on the operated side was good in 27.5 per cent, moderately good in 10.4 per cent, questionable in 1.8 per cent, and 1.8 per cent of the patients became worse after operation. Out of the remaining 53 patients (including 24 in whom no attempt was made to divide adhesions) whose cavities were not completely closed, or whose noncavernous lesions were not adequately collapsed, the effect on the operated side was excellent in 0.0 per cent, good in 0.0 per cent, mod-

erately good in 3.8 per cent, questionable in 28.3 per cent, unsatisfactory in 60.4 per cent, and 7.5 per cent of the patients became worse after operation. The corresponding percentages for the entire series of 111 patients are 30.6, 14.4, 7.2, 14.4, 28.9, and 4.5.

The operation was performed upon 92 patients who had cavities on the operated side. The cavities became completely closed in 51.1 per cent of the whole group; in 85.2 per cent of the 27 patients whose adhesions were completely divided, in 48.8 per cent of the 43 patients whose adhesions were incompletely divided, and in three, or 13.7 per cent, of the 22 patients who had no adhesions divided. In one of these three patients, closure followed a phrenic paralysis; in one, closure followed a pleural effusion that complicated operation; and in one, the continuation of an unaided partial pneumothorax resulted in cavity closure. If the last group of cases in which no adhesions were divided is excluded, 44, or 62.9 per cent, of the 70 patients whose adhesions were completely or partly divided had their cavities completely closed.

Closed intrapleural pneumonolysis was performed in 19 patients for noncavernous lesions, or for lesions that formerly included a cavity, in order to release the lesions from the intermittent tension produced by respiration through the adhesions. Pulmonary collapse was favorably affected in 92.9 per cent of the 14 patients whose adhesions were completely divided, in 33.3 per cent of the three patients whose adhesions were incompletely divided, and in neither of the two patients who had no adhesions divided. Pulmonary collapse was favorably affected in 73.7 per cent of the entire group of 19 patients.

The effect of the operation on the patient depends not only upon the operation's effect on the lung of the side operated upon, but also upon the subsequent behavior of lesions that may be present in the other lung, and upon the patient's general response to treatment. The results in our series appear in Table I. The chief reason for these results' being

TABLE I

RESULTS OF CLOSED AND OPEN METHODS OF INTRAPLEURAL PNEUMONOLYSIS

	111 PATIENTS* CLOSED METHOD PER CENT	29 PATIENTS OPEN METHOD PER CENT
Complete division of adhesions	37.0	65.5
Incomplete division	41.4	34.5
No division	21.6	0.0
Cavities closed	51.1†	55.2
Arrested or apparently arrested	20.7	20.7
Quiescent	14.4	3.4
Improved	42.3	55.1
Unimproved or worse	10.0	10.4
Dead	12.6‡	10.4§

*Of the 111 patients there were 92 with cavities and 19 with none.
†51.1% of the 92 patients with cavities.
‡Mortality directly or indirectly due to operation, 1.8%.
§Mortality directly or indirectly due to operation, 6.9%.

inferior to those reported for the effect of operation upon the lung and pleura of the operated side, is that the latter results are concerned only with the operated side. Among our 111 patients were 95, or 85.6 per cent, who had tuberculous infiltration of varying extent in the contralateral lung; these lesions were apparently active in 35 patients, and cavities were present in 31 of these 35. Various collapse therapy operations (the results of which were not included in the figures given in this paragraph) were used for the active lesions in the contralateral lung in most of the patients. The following 54 collapse therapy procedures also were later applied to the side of an unsuccessful closed intrapleural pneumonolysis operation in 41 patients: 21 open intrapleural pneumonolyses, 20 phrenic paralyses (in several instances to give protective relaxation and rest to a formerly extensively diseased lung after abandonment of the pneumothorax), 10 thoracoplasties, and three oleothoraces. The results of these procedures will not be reported in this article.

The operation produced a satisfactory effect upon the lung of the operated side in only three of the 14 patients who died; one of these three died from myeloid leucemia, one from myocardial degeneration a number of months after operation, and one from the effects of a pleuropulmonary perforation in a pneumothorax induced for contralateral lesions. The operation was technically unsuccessful in 11 of the 14 patients who died. In one of the 11 patients the tuberculosis before operation was classified as bilateral, far advanced C, with complicating ulcerative laryngeal tuberculosis. The patient was in poor condition, and the division of the adhesions over his huge cavity offered him his only chance for recovery; his heart was not known to be diseased, and a closed pneumonolysis operation was considered safe. The adhesions could be only incompletely divided. The patient developed extensive thoracic wall emphysema, cardiorespiratory decompensation, and died three days after operation. Another of the 11 patients died from a mixed infected empyema that succeeded a pure tuberculous empyema (this case has already been mentioned in this article). These two deaths are the only ones that were either directly or indirectly due to operation. The remaining nine patients died from progression of tuberculosis in one or both lungs; one of these patients had, in addition, severe diabetes, and another had tuberculous tracheobronchitis with progressive stenosis.

Open intrapleural pneumonolysis, or the division of adhesions through a wide surgical incision, was used by us in 29 cases from January, 1930, to October, 1934. In 21 of these cases, a previous closed intrapleural pneumonolysis operation had shown that the important adhesions were either too short or too complex to justify an attempt to divide them; in eight cases, the roentgenograms indicated that a closed operation would almost certainly be unsuccessful and, therefore, an

open operation was performed primarily. An anterior surgical approach was used in a few early cases and, thereafter, the far more efficient posterior approach.

Improvements in technique, contributed by several surgeons, have during recent years greatly reduced the danger that attended the type of operation that was used some years ago. In our 29 cases, bleeding from the divided surface of adhesions occurred four times; as the bleeding was immediately and easily controlled, it did not constitute a hemorrhage. The following postoperative complications, more than one of which occurred in each of several patients, developed:

1. Transient serous pleural effusion, 11 cases; a preoperative effusion had existed in two of these cases.

2. Persistent serous effusion, six cases. In one of these, a preoperative effusion had existed and tubercles were seen on the pleura during operation. In another patient, who had no preoperative effusion, tubercles were seen on the pleura. In three of the six patients, the serous effusion gradually developed into a pure tuberculous empyema. In a fourth patient, the fluid became slightly cloudy.

3. Partial loss of the pneumothorax from gradually progressive adhesive pleuritis, four cases.

4. Severe dyspnea, two cases. One of these occasionally had had some preoperative dyspnea, but none after operation until an effusion developed; the dyspnea was relieved by aspiration; the patient died suddenly one month after operation from a cardiac lesion. The other dyspneic patient, for whom the operation was performed to control repeated massive hemoptyses, was severely dyspneic during operation and intermittently so after operation until his death twenty-three days later; he had extensive tuberculous infiltration and cavitation in both lungs.

5. A temporary acute febrile reaction, a small subcutaneous pyogenic abscess, an extensive thoracic wall emphysema, and a temporary false hernial sac connecting a pure tuberculous empyema cavity and the deep layer of the thoracic wall, occurred in one case each. The acute febrile reaction was accompanied by a serosanguineous effusion which developed into a pure tuberculous empyema (listed under *2*) for which an oleothorax, and later a thoracoplasty, was successfully used. An adequate pneumothorax was maintained in the patient with extensive emphysema.

A moderate degree of thoracic wall emphysema developed in a majority of the patients, but with improvement in the technique of closure of the pleural incision, the amount of emphysema has been considerably reduced. No patient had a postoperative hemorrhage or sudden increase in the tuberculous lesions in either lung; none developed a mixed tuberculous and pyogenic empyema, a breaking open of the incision, or a pleurocutaneous sinus; none completely lost his pneumothorax invol-

untarily, and none died as a direct result of operation, although the operation was probably a contributing factor in the death of two patients (see *4*).

Each of the 29 cases* had one or more pulmonary cavities before operation, and almost all presented definite contraindication to thoracoplasty. The cavities became closed in 16, or 55.2 per cent, of the 29 cases, became smaller in 8, or 27.6 per cent, while the cavities were not affected in 5, or 17.2 per cent. A consideration of the supplementary operations which were used for the closure of cavities in the ipsilateral lung in several patients who had had unsuccessful pneumonolysis operations are not included in this report, except where the present condition of the patients is given on a following page. One patient had an oleothorax, five had phrenic paralysis, and five later became suitable for thoracoplasty.

The adhesions were completely divided in 19, or 65.5 per cent; in this group the cavities were completely closed in 73.7 per cent, became smaller in 15.8 per cent, and in two, or 10.5 per cent, the cavities were not affected by the operation, because in one patient the surrounding lung was exceptionally rigid, and in the other the pneumothorax was soon abandoned because of dyspnea. The adhesions were not completely divided in 10, or 34.5 per cent; in this group the cavities became closed in 20 per cent. At least some of the adhesions were divided in each of the 29 cases.

The pneumothorax was well maintained in 69 per cent of the 29 cases (in 73.7 per cent of the complete-division group, and in 60 per cent of the incomplete-division group), it was partially lost in 13.8 per cent, and was voluntarily abandoned in 17.2 per cent. The net effect of the operation on the operated side was excellent in 12, or 41.4 per cent, of the 29 cases, good in four, or 13.8 per cent, moderately good in five, or 17.2 per cent, poor, but no worse, in seven, or 24.1 per cent, and worse in one, or 3.5 per cent. Out of the 19 cases in which the adhesions were completely divided, excellent, good, and moderately satisfactory results were obtained on the operated side in 89.4 per cent, including 57.8 per cent in the excellent category. Out of the 10 cases in which the adhesions were incompletely divided, excellent, good, and moderately satisfactory results were obtained in 40 per cent, including 10 per cent in the excellent category.

The present clinical condition of the patients is of interest, although in several of them supplementary operations (as noted in the following paragraphs) became necessary on the side of the pneumonolysis op-

*There were actually only 28 patients, but since one patient had bilateral operations he must be considered as two cases. The cavity on the side first operated upon closed within a month. The other side was operated upon six months after the first operation; some of the adhesions could not be divided, the pneumothorax was abandoned a month later, and a staged seven-rib thoracoplasty performed two months later. A year after the thoracoplasty no cavity could be seen in roentgenograms, but the sputum was occasionally positive.

eration. The patients were followed from four months to nearly three years after operation. The results, according to the classification of the National Tuberculosis Association, are given in Table I. Two of the six patients classified as arrested, or apparently arrested, had a supplementary phrenic paralysis and thoracoplasty on the side of the pneumonolysis operation. The patient classified as quiescent is a negress who had bilateral cavitation and bilateral pneumothorax.

Ten of the 16 patients who are classified as improved had complete closure of the cavity on the operated side, to which a phrenic paralysis operation was added in two patients. Three of the 10 had minimal tuberculosis in the contralateral lung, for which a temporary phrenic paralysis was used in two. One of the two patients with moderately advanced tuberculosis in the contralateral lung had a temporary phrenic paralysis bilaterally. The five patients who had far-advanced tuberculosis in the contralateral lung had the following operations for these lesions: (1) temporary phrenic paralysis; (2) and (3) pneumothorax; (4) pneumothorax, closed intrapleural pneumonolysis, and temporary phrenic paralysis; (5) pneumothorax succeeded by thoracoplasty. A number of the 16 improved cases, as well as the one quiescent case, will probably be entitled to an arrested, or apparently arrested, classification after the required postoperative period has passed. Among the six improved patients whose open pneumonolysis operation was unsuccessful in completely closing the cavities, two had minimal tuberculosis in the contralateral lung; a thoracoplasty was eventually required on the pneumonolysis side in one of them. Four of the improved patients had far-advanced tuberculosis in the contralateral lung; for two of these patients, pneumothorax and closed intrapleural pneumonolysis were used, and for one, only pneumothorax. Two of the four patients with bilateral far-advanced tuberculosis eventually became fit for thoracoplasty on the side of the open pneumonolysis operation, one of them having had oleothorax and phrenic paralysis before the thoracoplasty was performed; a third patient is now ready for thoracoplasty.

Pneumonolysis was technically unsuccessful in the three patients classified as unimproved or worse; one had moderately advanced and two had far-advanced tuberculosis in the contralateral lung. One of them is perhaps slightly improved, rather than unimproved, and may become suitable for further surgery; this patient developed a pure tuberculous empyema and a false hernia of the thoracic wall, both of which disappeared without surgical treatment. The second patient has had a thoracoplasty on the contralateral side, but too recently for an estimation of the result. The third patient has had gradual progression of bilateral lesions, and a preoperative pleural effusion persists.

Three of the 29 patients have died. The circumstances of the death of two of the three have already been reported in this article. The

third patient did well until five months after the pneumonolysis operation when she developed renal colic, was operated upon for a calculus, and died several days later from a cerebral complication.

One hundred of our patients had cavities for which the closed or open types of pneumonolysis operation, or both, were used. The cavities became closed in 63 per cent. This figure applies to all the patients who had cavities, whether the adhesions were completely or incompletely divided, or not divided at all.

CONCLUSIONS

Closed intrapleural pneumonolysis is a highly valuable operation for certain carefully selected cases of inadequate pneumothorax that are unsuitable for treatment by phrenic paralysis. In well-selected cases, closed pneumonolysis is decidedly preferable to thoracoplasty. Furthermore, in a considerable proportion of patients for whom closed pneumonolysis is technically possible, thoracoplasty is contraindicated because of too active lesions in the contralateral lung, or for other reasons.

Pneumonolysis is not a definitive operation. Its only purpose is to produce a satisfactory pneumothorax. The patient is, therefore, exposed to the double danger of the potential complications of both pneumonolysis and pneumothorax. The danger is, however, small and the results are excellent when the adhesions that need to be divided are clearly suitable for the operation.

Open intrapleural pneumonolysis is valuable for a small group of patients whose adhesions are too short or complex in arrangement for safe division by the closed method of pneumonolysis. An open operation may be used for certain cases of bilateral active tuberculosis for which a thoracoplasty would be definitely contraindicated. However, the magnitude of the open operation, and its potential dangers, make it less desirable than thoracoplasty for patients who present no contraindication to thoracoplasty. We believe that open intrapleural pneumonolysis should be reserved for those patients in whom phrenic paralysis, if indicated, and closed intrapleural pneumonolysis have already been tried, and in whom thoracoplasty is contraindicated, and to whom the open operation offers the only chance of recovery.

ARTIFICIAL PNEUMOTHORAX: WITH PARTICULAR REFERENCE TO THE AMBULATORY PATIENT*

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IN THE treatment of pulmonary tuberculosis there are three main objectives: the first, to check the spread of the disease and the breaking down of the tissues in the individual patient at the earliest possible moment; the second, to prevent the lesion from disseminating tubercle bacilli to others, or stop such dissemination if it is already occurring when the disease is diagnosed; the third, to continue the working capacity of the patient, or to restore him in the shortest possible time consistent with the safety of his health and associates. To attain these objectives, collapse therapy is more effective than all the other methods employed throughout the ages. The less extensive the pulmonary involvement when treatment is begun, the better the prognosis, generally speaking.

We now have sufficiently refined diagnostic aids to detect chronic pulmonary tuberculosis of the destructive type long before symptoms appear. When found in the presymptom stage and determined to be progressive, collapse therapy has its most valuable place both from the standpoint of the patient and the community. Unfortunately, our refinements in diagnosis are not yet so generally used as to detect all cases of pulmonary disease in the presymptom stage. There are still persons, and many of them, who have their tuberculosis diagnosed only a short time before death. I have recently seen a man who was very active in his work, but whose first examination revealed far advanced chronic tuberculosis involving both lungs. In fact, he died of this disease within ten days of the examination. For such persons, we have nothing to offer except treatment of a palliative nature, but for their families, we may stop further exposure by isolating the patient at the earliest possible moment after the disease is detected.

Fortunately, between the minimal stage when so much can be done, and the far advanced bilateral stage when there is so little to do, there are many steps in the development of the disease when collapse therapy is of avail.

Various methods of bringing about rest or collapse of the diseased lung, such as weights on the chest wall, interruption of the phrenic

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nerve, artificial pneumothorax, and extrapleural thoracoplasty, have proved their worth. Artificial pneumothorax, however, is the most popular of all the methods and should be the first method attempted whenever collapse is indicated, because a very satisfactory collapse can usually be accomplished if the lung is not adherent to the chest wall, and, moreover, one has considerable control over the lung and can later allow it to re-expand when the parts not previously destroyed by disease, resume their normal function. In addition, artificial pneumothorax is easy to administer.

Since many of the published reports on the results of artificial pneumothorax treatment have come from institutions where the majority of the patients had the treatment instituted only after the disease was moderately or far advanced, artificial pneumothorax has been looked upon by many as a procedure for pulmonary tuberculosis in the more advanced stages of the disease. Moreover, since in the past, approximately 80 per cent of patients with pulmonary tuberculosis had their disease diagnosed only after it was well advanced, largely because the symptoms are slight or absent before this stage, there has not been much opportunity to institute artificial pneumothorax for minimal disease. This has led to statements, such as that of Coryllos,[1] to the effect that in artificial pneumothorax work there are 70 to 80 per cent failures or deaths. However, with our present methods of case-finding among apparently healthy persons, the number found with minimal disease is very definitely increasing. Obviously, it is better to treat a tuberculous process by artificial pneumothorax before cavities and adhesions have formed, before large volumes of lung tissue have been involved, and before the patient's general health has become undermined, than to wait until these conditions have developed.

In 1935, a report[2] was made on fifty-two patients with pulmonary tuberculosis, whose treatment was instituted while the disease was minimal. In these patients, however, the disease was determined to be definitely progressive before treatment was instituted. Of the fifty-two, only three were found to have adhesions sufficiently extensive to interfere with collapse. At the time the report was made, forty-two of the group were working or ready to work; four had died, one of whom had miliary tuberculosis, another multiple lesions in various parts of the body, and the other two, nontuberculous conditions. The results in this group of patients were "superior to those in other groups under our care, that had artificial pneumothorax instituted after the disease had become moderately or far advanced. They are also superior to those of other groups of minimal cases which we have observed on bed rest treatment alone." At that time, attention was called to the results obtained by a number of other workers[3-14] who had employed or were recommending artificial pneumothorax for early cases of tuberculosis.

Turner and Collins[15] made observations on patients with minimal pulmonary tuberculosis treated by various methods and found "that the watchful waiting policy for evidence of progression proved costly and dangerous in several cases, and even fatal in a few. Therefore, we advocate immediate induction of artificial pneumothorax in definitely diagnosed Minimal cases." These authors have not seen a "Minimal pneumothorax case show progression of the disease in the treated lung, or metastasis to a new lobe or exhibit an exacerbation of a previously existing lesion in the contralateral lung." They have found that those patients who received at least six months' rest treatment after pneumothorax was induced did better than those for whom this was not possible. They state, however: "As we are more certain of our results we may consider it feasible to allow these people to return to work long before their course of pneumothorax treatment has been completed." After a careful study of 139 patients with minimal disease treated by various methods, Turner and Collins conclude: "The operative risk in Minimal cases is negligible. Percentage of cases with free space is high in Minimal cases. The high percentage of effective collapses in Minimal pneumothorax cases is a guarantee of good results. Complications were so infrequent that they should not constitute a deterrent for the use of pneumothorax in early cases. The conversion of sputum in positive Minimal cases is prompt and certain under pneumothorax treatment. The conversion time, hospital stay and total disability were definitely decreased by the use of pneumothorax. Pregnancy in Minimal cases is well tolerated if supported by pneumothorax.* The conservative treatment of Minimal pulmonary tuberculosis requires the early induction of artificial pneumothorax. We believe that the existence of a Minimal tuberculous lesion in an individual of 'Teen' age is an absolute indication for artificial pneumothorax without delay."

In 1933, Lawrason Brown[16] said: "It is highly likely that in the future artificial pneumothorax will be induced earlier in many instances. Economically important, such a method would no doubt return many patients far sooner to work for it would hasten the disappearance of tubercle bacilli in the sputum. The length of residence in the sanatorium or hospital would be shortened. It is cheaper for any community to establish 'artificial pneumothorax' clinics than to maintain patients over long periods of time in sanatoriums."

Since many of the reports on artificial pneumothorax therapy have come from institutions where patients have been given long periods of bed rest, in addition to collapse therapy, it has been difficult to determine how much credit for the success obtained should be given bed rest and how much to collapse therapy. However, there are now groups of patients on record where treatment was limited to sanatorium treatment

*Four of the cases receiving pneumothorax carried a pregnancy to term without any apparent ill effects.

or strict bed rest, without the use of collapse therapy, and others, where treatment was largely limited to collapse therapy. For example, Wherrett[17] observed, for periods of one to thirteen years, 291 patients with minimal tuberculosis, who had only sanatorium treatment. In 28 per cent, the results were not good. Pope[18] has reported thirty-four patients in the "teen"age period who had minimal disease when admitted to the sanatorium. Four to seven years later, 40 per cent were dead.

In 1933, a report[19] was made on sixty-six patients with pulmonary tuberculosis who had been treated by ambulatory artificial pneumothorax. Thirty-two of these patients were given periods of bed rest, varying from a few days to three months, while artificial pneumothorax was being instituted. The remaining thirty-four were entirely ambulatory. Attention was called to the fact that the institution of artificial pneumothorax for the ambulatory patient is not a new procedure in this country, since it was employed by Tice in 1898. Although in 1933 it had not been extensively used, reference was made to the work of several authors[20-26] who in recent years had instituted artificial pneumothorax for patients who remained ambulatory, or had only brief periods of strict bed rest at the beginning of the treatment. This procedure is fast gaining ground among the chest specialists.

Douglas[27] calls attention to the program in Detroit where patients are hospitalized until artificial pneumothorax has resulted in closure of cavities and negative sputum, after which they are discharged from institutions and treated as ambulatory patients. The present policy at the Minnesota State Sanatorium[28] consists of instituting artificial pneumothorax and returning the patient to the private physician soon after the sputum has become negative. Physicians who have not previously had experience in artificial pneumothorax work are taught the procedure.

Haygood[29] has described the program in Georgia, where he points out that maintenance of patients on artificial pneumothorax treatment in the sanatorium costs the state $18.60 for each treatment. He says: "This work, we reasoned, could be done by the family physician if he were trained in the technique of pneumothorax operation." The Georgia program consists of returning patients to their private physicians for continuation of artificial pneumothorax treatments at the earliest possible moment. Obviously, by these two state sanatorium methods, large numbers of beds are made available for patients with communicable tuberculosis whose disease cannot be controlled by collapse therapy and who really need institutional care. Among the physicians engaged in private practice, ambulatory pneumothorax is in wide use but few of these physicians have reported their cases in the literature.

Cole and Harper[30] have presented their experience with ambulatory artificial pneumothorax which began prior to 1925, because of dearth of beds for tuberculous patients. The patients in whom artificial pneu-

mothorax was indicated were admitted to a general hospital while the treatment was being instituted. After more than ten years of experience with ambulatory pneumothorax, they say:

"We soon discovered, much to our amazement, that many of these patients whom we treated in a general hospital for a short period of time while deflating the lung became symptom-free so promptly, although coming to our offices for refills while awaiting their turn for admission to a sanatorium, that we frequently found it difficult and sometimes even impossible to convince such patients of the need for sanatorium care. We were skeptical, at first, about the ultimate outcome of these patients who through necessity were receiving this ambulatory treatment, but as time went on we found that they continued to do as well, and some of them better, than other similar patients who had the advantage of prolonged sanatorium care. We found further that the patients who were hospitalized for a short period of time and returned home were able to resume work much sooner and had much less readjustment to undergo than similar patients who had remained in sanatoria so long as to become estranged. In many instances, we found it more difficult to rehabilitate the patient who had been in an institution for months or years than the patient who had been hospitalized for a short period of time during the institution of pneumothorax and then permitted to return home, coming to the office for refills. At first we did not permit these patients to return to work until months had elapsed, but we gradually shortened this period of idleness until now most patients are back at work within a few months, many of them within a few weeks, some of them within a few days, and a few of them never stop work."

Cutler and others[31] found ambulatory artificial pneumothorax very satisfactory among negro patients.

Edlin and Tannenbaum[32] have found ambulatory treatment satisfactory among patients who require bilateral artificial pneumothorax.

In Chicago, Tice and Hruby[33] reported 626 dispensary patients received ambulatory artificial pneumothorax in 1935. They are enthusiastic over the results and possibilities of this form of treatment for such reasons as conversion of positive to negative sputum, thus stopping the spread of tubercle bacilli, and continued or quick restoration of the working capacity of the individual patient. In the summary of their entire work, which includes approximately seven hundred persons with tuberculosis whose sputum has been rendered negative, they say: "It must be stated too, that the approximately 700 open cases closed, came, practically without exception, from the congested districts, from crowded, unhygienic homes, in many of which were children or young adolescents. We have no need to strain our imagination to realize the significance of this. Seven hundred fountains of infection damned at

the source, a town of seven hundred carriers walled off behind the ramparts of collapse must be conceded an increasingly tangible influence on mortality and morbidity rates.''

The patients reported in this paper have in no way been selected. They are presented just as they reported for treatment and are grouped as follows: Group I consists of 239 patients with unilateral disease, who have at any time been ambulatory while receiving artificial pneumothorax treament, and sixty patients with bilateral disease when treatment was begun or later developed significant lesions in the contralateral lung and became ambulatory at some time during treatment. Group II consists of thirty-five patients who did not become ambulatory while receiving artificial pneumothorax. Group III consists of more than one hundred patients treated for a short period of time, such as transients and those whose treatment was instituted for other physicians. Group IV consists of ninety-two patients for whom artificial pneumothorax was recommended but found impossible because of adhesions.

Two hundred thirty-nine of the patients in Group I had lesions confined to one lung, so far as could be determined by examination, including x-ray film. These patients have been divided into two main subgroups: The first consists of those who were on strict bed rest three months or less during and immediately following the beginning of artificial pneumothorax. The second subgroup is comprised of those patients who were on strict bed rest for more than three months during and immediately following the institution of artificial pneumothorax.

TABLE I

	STRICT BED REST 3 MONTHS OR LESS		STRICT BED REST MORE THAN 3 MONTHS			
	NO. OF CASES	PER CENT OF TOTAL	NO. OF CASES	PER CENT OF TOTAL	TOTAL CASES	PER CENT OF TOTAL
Min.*	42	35.3	16	13.3	58	24.3
M. A.*	54	45.3	70	58.3	124	51.9
F. A.*	23	19.4	34	28.4	57	23.8
	119	100.0	120	100.0	239	100.0

*Min., minimal; M. A., moderately advanced; F. A., far advanced.

This division was made as it was desired to determine, with as much accuracy as possible, whether there is any significant difference in the two groups, with reference to the development of complications subsequent to the beginning of collapse by artificial pneumothorax, the number for whom other collapse therapy procedures became necessary, the working capacity, and the number who died of tuberculosis. One hundred and nineteen received three months or less of strict bed rest during and immediately following the institution of artificial pneumothorax. Of the 119 patients, 35.3 per cent had minimal, 45.3 per cent moderately advanced, and 19.4 per cent far advanced disease when artificial pneumothorax was instituted. The remaining 120 of the 239 patients

had, in addition to artificial pneumothorax, strict bed rest over periods exceeding three months, some of them for several years. Of these patients, 13.3 per cent had minimal, 58.3 per cent moderately advanced, and 28.4 per cent far advanced disease when artificial pneumothorax was begun.

The age distribution of these patients is shown in percentage in Table II, where it will be observed that by far the largest number is between 20 and 29 years.

TABLE II

	STRICT BED REST 3 MONTHS OR LESS			STRICT BED REST MORE THAN 3 MONTHS		
AGE	MIN.	M. A.	F. A.	MIN.	M. A.	F. A.
10-19	23.8	11.1	4.3	18.8	10.0	11.8
20-29	59.5	57.4	56.5	62.5	60.0	44.1
30-39	16.7	22.2	34.8	18.8	24.3	35.3
40-49		1.8	4.3		2.8	8.8
50-59		3.7			1.4	
60-69		3.7			1.4	

Table III shows the percentage of patients in each stage of the disease with partial and complete pneumothorax. Eighty-one per cent, 51.8 per cent, and 47.8 per cent, respectively, of the patients with minimal, moderately advanced, and far advanced disease who were on strict bed rest three months or less had complete pneumothorax, whereas, the collapse was complete in 68.8 per cent, 60 per cent, and 64.7 per cent, respectively, of the minimal, moderately advanced, and far advanced cases, who received more than three months of strict bed rest.

TABLE III

	STRICT BED REST 3 MONTHS OR LESS			STRICT BED REST MORE THAN 3 MONTHS		
Pneumothorax	MIN.	M. A.	F. A.	MIN.	M. A.	F. A.
Partial	19.0	48.2	52.2	31.2	40.0	35.3
Complete	81.0	51.8	47.8	68.8	60.0	64.7

The percentage of patients who have had various complications with pneumothorax is indicated in Table IV.

TABLE IV

	STRICT BED REST 3 MONTHS OR LESS			STRICT BED REST MORE THAN 3 MONTHS		
Complications	MIN.	M. A.	F. A.	MIN.	M. A.	F. A.
Empyema		5.5	17.3	6.2	4.2	11.8
Emboli		1.8				
Accidental pneumothorax						5.8

Empyema is usually looked upon as a fairly frequent complication. Most workers agree that it is rarely produced by pathogenic microorganisms, reaching the pleural cavity through the needle, but that

nearly all cases, whether they are due to tubercle bacilli or to mixed infections, are the result of the escape of microorganisms from the foci of disease previously involving the lung or pleura. The giving way of an adhesion at its attachment to the visceral pleura may suffice in tearing the pleura and even the lung tissue to such an extent that infection escapes from the lung into the pleural cavity. Again the disease may burrow through the lung tissue and visceral pleura and discharge microorganisms into the pleural cavity. The rupture of a thin wall of a pulmonary cavity is not an uncommon source of pathogenic microorganisms in cases of empyema. When tubercle bacilli are the sole cause, they probably are usually transmitted by way of the lymphatics to the visceral pleura in a manner similar to that so frequently seen in the development of simple tuberculous pleural effusions.

Among the forty-two patients who had artificial pneumothorax instituted while the disease was minimal and were on bed rest treatment three months or less, none has developed empyema. Among the sixteen patients with treatment instituted for minimal tuberculosis but who were also treated by strict bed rest, three months or more, one has developed empyema. This girl had treatment instituted in June, 1927, after which she spent approximately one year in bed. Following a course of graduated exercise, she returned to her work and continued with an excellent working capacity until April, 1933, when she developed mixed infection empyema on the side of the artificial pneumothorax with a small pleuropulmonary fistula.

Among the patients with moderately advanced disease who had three months or less of strict bed rest at the time artificial pneumothorax was instituted, three (5.5 per cent) developed empyema. Each of them had only tuberculous empyema. In one case, sterile mineral oil was introduced to replace the air and pus. This patient was not confined to bed, since there were no significant symptoms present. The oil has now been removed, and the lung has completely re-expanded. Another patient had oil substituted for air, and this treatment has been continued for more than a year, with the patient on full working capacity. For the third patient who developed tuberculous empyema, pus was aspirated and later the lung was allowed to re-expand. The empyema developed in November, 1931, and the re-expanded lung now appears to be in excellent condition.

Three (4.2 per cent) of the patients with moderately advanced disease who had strict bed rest more than three months after pneumothorax was instituted, developed empyema. One remained in an institution for approximately twenty-two months during which time the lung expanded, and for the past five years she had been in good health. Another, whose empyema developed in 1926, remained in the institution for twenty-eight months, during which time extrapleural thoracoplasty was performed. She now has a good working capacity. The third had

empyema develop in 1930. The same year extrapleural thoracoplasty was performed. This man has not yet been restored to working capacity.

Among the twenty-three patients with far advanced disease who had three months or less of strict bed rest when artificial pneumothorax was begun, four (17.3 per cent) have developed empyema. Two of these patients had only tuberculous empyema and were treated by aspiration, and the lungs later re-expanded with good result. A third patient has had rib resection and is now being prepared for extrapleural thoracoplasty. This woman had been treated in a sanatorium for twelve months, 1927-28, after which she left the institution. Pneumothorax was not begun until 1931, and the empyema developed in 1934. The fourth patient had a large tuberculous cavity in an upper lobe which could not be collapsed by artificial pneumothorax and developed a pleuropulmonary fistula with mixed infection empyema. This patient is now being prepared for extrapleural thoracoplasty.

Four (11.8 per cent) of the patients who had far advanced disease with more than three months of strict bed rest after artificial pneumothorax was begun, have developed empyema. In one case, the pus was aspirated and the pneumothorax treatment continued. This was approximately four years ago, and the patient has remained in good health. Another patient with tuberculous empyema has had sterile mineral oil introduced into the pleural cavity without any loss of working capacity. The two remaining patients required thoracoplasty. In one, the empyema developed in 1931, and the patient was able to return to work in 1936. In the other, the empyema developed in 1926, and the patient returned to work in 1929 and has since had good working capacity.

In the entire group of 239 patients, gas embolism occurred in only one. Symptoms were present over a period of two or three days, after which the artificial pneumothorax was continued. Two patients (5.8 per cent) developed what we believed to be spontaneous pneumothorax in the opposite lung. Both of these patients had far advanced disease and had been treated over long periods of time on strict bed rest. Both, however, were ambulatory at the time the complication developed. Death occurred within a few minutes after symptoms appeared.

Table V shows the percentages of patients who have had other special methods of treatment administered in addition to artificial pneumothorax.

TABLE V

	STRICT BED REST 3 MONTHS OR LESS			STRICT BED REST MORE THAN 3 MONTHS		
	MIN.	M. A.	F. A.	MIN.	M. A.	F. A.
Phrenic interruption		1.8	17.3		12.8	17.6
Intrapleural pneumonolysis			4.3			
Oleothorax		3.7	8.6		1.4	2.9
Thoracoplasty	2.3	1.8	8.6		4.2	11.8

It will be observed that none of the patients who had pneumothorax instituted for minimal pulmonary tuberculosis required interruption of the phrenic nerve.

Among the patients with moderately advanced disease who had three months or less of strict bed rest, one (1.8 per cent) had the phrenic nerve interrupted. This was at the termination, because of adhesions of the artificial pneumothorax. Among those who had more than three months of strict bed rest, nine (12.8 per cent) of the moderately advanced cases had the phrenic nerve interrupted. In seven of these cases, this operation was performed because obliterative adhesions were developing, or had caused the complete loss of the pleural space. In two, this operation was performed in an attempt to obtain a better collapse.

Among the patients with far advanced disease who had three months or less of strict bed rest, four (17.3 per cent) have had interruption of the phrenic nerve, three of whom were losing their collapse through the formation of obliterative adhesions. For the fourth, this operation was performed in an attempt to secure a better collapse of the lung. Among the patients with far advanced disease who were treated by strict bed rest for more than three months after artificial pneumothorax was instituted, six (17.6 per cent) have since had interruption of the phrenic nerve. In three of these cases, the operation was performed after the lung had re-expanded. In two, it was performed preparatory to extrapleural thoracoplasty, and in one, in an attempt to secure a better collapse.

In the entire group of 239 patients, intrapleural pneumonolysis was performed for only one (4.3 per cent) who was a case of far advanced pulmonary tuberculosis, classified with those who had three months' or less strict bed rest. Her open cavity closed after cauterization of the adhesion.

Oleothorax was substituted for pneumothorax in none of the minimal cases. Among the patients with moderately advanced disease who received three months or less of strict bed rest, two (3.7 per cent) have had this treatment instituted because of tuberculous empyema. It did not, however, interfere with the working capacity in either case. One patient (1.4 per cent) with moderately advanced tuberculosis who had had more than three months of strict bed rest had oil introduced into the pleural cavity because obliterative adhesions were developing.

Among the patients with far advanced disease who had three months or less of strict bed rest, oleothorax was instituted for one because of obliterative adhesions, and another in an attempt to establish a more uniform collapse of the lung (8.6 per cent). Among the patients with far advanced disease who had more than three months of strict bed rest, one (2.9 per cent) had oleothorax instituted because of tuberculous empyema.

Only one patient (2.3 per cent) from the group with minimal disease who had strict bed rest for three months or less, later came to thoracoplasty. This patient had a very satisfactory collapse by artificial pneumothorax. However, approximately eighteen months after it was instituted, she was examined by a physician in another state who strongly recommended that the pneumothorax be discontinued. This was done, and slightly more than two years later, the disease had reactivated and extended so as to require more drastic treatment. None of the patients with minimal disease who had more than three months' strict bed rest were later treated by thoracoplasty.

Among the patients with moderately advanced disease who had three months or less of strict bed rest, one (1.8 per cent) later required extrapleural thoracoplasty. This patient had only a partial collapse which was not adequate to control the disease. Among those with moderately advanced disease who had more than three months of bed rest, three (4.2 per cent) later required thoracoplasty. Two, because of empyema, and a third, because the artificial pneumothorax was lost through obliterative adhesions.

Of the patients with far advanced disease who had three months or less of strict bed rest, two (8.6 per cent) have since required extrapleural thoracoplasty, one because of empyema, and the other because of loss of the pneumothorax space through obliterative adhesions. Of those with far advanced disease who received more than three months of strict bed rest, four (11.8 per cent) have since required extrapleural thoracoplasty. Two because of empyema, one because of a bloody effusion which persisted, and one because of reactivation of disease after artificial pneumothorax had been lost through obliterative adhesions.

In Table VI the percentage of patients who have discontinued pneumothorax for various reasons is shown for each stage of disease.

TABLE VI

	STRICT BED REST 3 MONTHS OR LESS			STRICT BED REST MORE THAN 3 MONTHS		
	MIN.	M. A.	F. A.	MIN.	M. A.	F. A.
Adhesions	10.0	22.0	22.0	31.0	30.0	9.0
Recommendations and voluntary	26.0	5.0	13.0	19.0	18.0	21.0
Empyema		4.0	9.0	7.0	3.0	9.0
Died tuberculosis		2.0				8.0
Died other causes		2.0	4.0	6.0		3.0
No reason given				6.0		
Not discontinued	64.0	65.0	52.0	31.0	49.0	50.0

While a lung is collapsed, it is impossible to determine when tuberculous lesions are sufficiently controlled to make it safe to allow the lung to re-expand. Much depends upon the extent of disease when treatment is begun, as well as the degree of collapse one has been able to maintain. Many workers are of the opinion that the lung should be kept under a state of collapse for at least two or three years for minimal

or early moderately advanced lesions, and for a longer period of time for late moderately advanced and far advanced lesions. The question most frequently asked by patients on artificial pneumothorax treatment is regarding the time of discontinuing treatment. Because they are entirely free from symptoms, some patients assume the responsibility and voluntarily discontinue. The majority, however, are willing to wait for the physician's recommendation.

Unfortunately, one is not always able to continue artificial pneumothorax as long as it seems necessary or as the patient desires. Of the reasons for discontinuing treatment prematurely, obliterative adhesions which often follow serous effusions is far in the lead. Also when adhesions are already present to such an extent as to prevent complete collapse of the lung at the time artificial pneumothorax is instituted, it is not uncommon to see adhesions slowly fill the pleural space. When obliterative adhesions begin to form, increasing the air pressure in the pleural cavity usually has little effect in preventing their further development. However, the introduction into the pleural cavity of sterile mineral or olive oil so as to replace the greater part of the air will often suffice to keep the lung under a remedial state of collapse. A good many of the patients here reported lost their collapse by obliterative adhesions before the use of oil was known to be valuable.

In Table VI it will be observed that among those patients who had three months or less of strict bed rest, 64 per cent, 65 per cent, and 52 per cent of the minimal, moderately advanced, and far advanced cases, respectively, are still on treatment; while of those who had strict bed rest more than three months, 31 per cent, 49 per cent, and 50 per cent of the minimal, moderately advanced, and far advanced cases, respectively, have not discontinued the treatment.

In Table VII the patients have been grouped with reference to discontinuing work after having become ambulatory, and according to survival.

TABLE VII

	STRICT BED REST 3 MONTHS OR LESS			STRICT BED REST MORE THAN 3 MONTHS		
	MIN.	M. A.	F. A.	MIN.	M. A.	F. A.
Stopped work	7.1	5.5	21.7	12.5	15.7	14.7
Died tuberculosis	2.3	1.8	4.3		4.2	14.7
Died other causes	2.3	1.8	4.3	6.2	1.4	2.9

Among the patients with minimal disease who had three months or less of strict bed rest, three (7.1 per cent) stopped work and entered hospitals. One of them had done well on ambulatory artificial pneumothorax for two years, but an older sister was convinced that no one recovers from tuberculosis without a period of sanatorium treatment. With no reason for hospitalization whatsoever, except the opinion of the sister, this girl was forced to enter an institution, where she remained

approximately five months, after which she resumed her activities. Another girl who was treated by ambulatory artificial pneumothorax lost her collapse through formation of obliterative adhesions at the end of approximately one year. As the lung re-expanded, the thickened pleura cast a homogeneous shadow which appeared very significant to the family physician. This, together with occasional chest pain, caused the physician, who had not previously treated the lung condition, to recommend that she stop work immediately and enter a sanatorium. In neither of these cases was there any true reason why work should have been discontinued. The third patient, after eighteen months of ambulatory treatment, was advised by a physician in another state to discontinue artificial pneumothorax. She had a good collapse of the diseased lung, and if it had been continued, it seems highly probable that she would not have found it necessary to discontinue work, enter a hospital, and have extrapleural thoracoplasty performed.

Among the patients with minimal disease who had more than three months of strict bed rest, two (12.5 per cent) have stopped work. One was a girl who was reacting satisfactorily and had no symptoms, but relatives insisted that she discontinue work and enter a sanatorium. This was entirely unnecessary. The second, because of a pleuropulmonary fistula and empyema, was forced to discontinue work and enter an institution.

Three (5.5 per cent) patients with moderately advanced disease who had three months or less of strict bed rest have stopped work. One because of a large serous effusion with rather severe symptoms became alarmed and asked to be admitted to a sanatorium. She remained in the institution ten months, then returned to her usual activities, and has since continued on ambulatory artificial pneumothorax. Another patient had collapse that was not sufficient to control his disease, so he was advised to discontinue work, enter an institution, and have thoracoplasty performed. The third patient lost her collapse through the development of obliterative adhesions and her disease reactivated to such an extent that she was advised to discontinue work, and is now considering extrapleural thoracoplasty.

Eleven (15.7 per cent) patients with moderately advanced tuberculosis who had received more than three months of strict bed rest later discontinued work. For only one of the eleven was empyema the cause. This patient returned to a sanatorium and later had extrapleural thoracoplasty performed. Two patients voluntarily discontinued artificial pneumothorax strongly against advice. Both of them later reactivated. One has died and the other has the disease in a hopelessly advanced stage. One patient, who was doing well, was advised by friends to enter a sanatorium where he remained for about four months, after which he resumed his activities. Obliterative adhesions caused the loss of collapse in three cases, after which the disease

reactivated. Two have returned to the sanatorium, and one died at the age of seventy-five years. Three have had extension of the disease to other parts of the body, one of whom has died, and two have the disease in a hopelessly advanced stage. One patient has stopped work and returned to the sanatorium on two occasions for brief periods, once for two months, and again for one month.

Among the patients with far advanced disease who had strict bed rest over a period of three months or less, five (21.7 per cent) stopped work and returned to or entered sanatoriums. Two lost their collapse through the development of obliterative adhesions, and the disease later reactivated. One developed empyema and a new lesion in the opposite lung; one a pleuropulmonary fistula with mixed infection empyema. The fifth was advised to enter an institution long enough to have intrapleural pneumonolysis performed. Following this, however, a large pleural effusion developed, and she was retained in the institution for more than a year. The lung is now satisfactorily collapsed, and this patient is engaged in the usual activities of life. Among the patients with far advanced disease who had more than three months of strict bed rest, five (14.7 per cent) discontinued work. One voluntarily discontinued pneumothorax treatment and later had reactivation of the disease, and it spread to the opposite lung. Another lost her collapse through obliterative adhesions and later required extrapleural thoracoplasty. Another developed a bloody effusion, when she was advised to discontinue work and later to have extrapleural thoracoplasty. One had an unsatisfactory collapse and a rapid spread of the disease to the contralateral lung, which caused death in a short time. The fifth patient died of miliary tuberculosis.

At the beginning of the fifth year interval (Table VIII), one sees that all of the patients with minimal tuberculosis who were on strict bed rest three months or less were alive. However, subsequently two have died (Table VII), one in a highway accident and one of miliary tuberculosis, eight years after artificial pneumothorax was discontinued. At the beginning of the fifth year interval (Table VIII), 93.8 per cent of the patients with minimal disease, who had strict bed rest more than three months, were alive. One died of nontuberculous pneumonia, while still on artificial pneumothorax treatment (Table VII).

At the beginning of the fifth year interval (Table VIII), 98.2 per cent of the patients with moderately advanced tuberculosis who had been treated by strict bed rest three months or less were alive; one died of malignancy while the tuberculosis was well controlled; another, who had an unsatisfactory collapse, had reactivation and rapidly progressive tuberculosis in the contralateral lung, died subsequent to this period. At the beginning of the fifth year interval (Table VIII), 96.9 per cent of the patients with moderately advanced disease who had

strict bed rest more than three months immediately following the institution of artificial pneumothorax were alive. One died of tuberculosis. Subsequent to this period, three have died; one of nontuberculous pneumonia fourteen months after artificial pneumothorax had been discontinued, and she had been restored to a full working capacity as far as tuberculosis was concerned. The remaining two died after pneumothorax had been discontinued, when the disease reactivated and extended to other parts of the body.

TABLE VIII

SURVIVAL RATE AFTER PNEUMOTHORAX STARTED AT BEGINNING OF EACH SUCCEEDING YEARLY INTERVAL

INTERVAL	STRICT BED REST THREE MONTHS OR LESS						STRICT BED REST MORE THAN THREE MONTHS					
	MIN.		M.A.		F.A.		MIN.		M.A.		F.A.	
	NO. PTS.	% ALIVE	NO. PTS.	% ALIVE	NO. PTS.	% ALIVE	NO. PTS.	% ALIVE	NO. PTS.	% ALIVE	NO. PTS.	% ALIVE
0-1	42	100	54	100.0	23	100.0	16	100.0	70	100.0	34	100.0
1-2	31	100	44	98.2	20	100.0	16	100.0	70	100.0	33	97.1
2-3	24	100	37	98.2	15	90.0	16	100.0	68	100.0	32	94.0
3-4	17	100	26	98.2	13	90.0	15	93.8	64	100.0	26	90.9
4-5	14	100	17	98.2	8	90.0	12	93.8	56	96.9	21	90.9
5-6	9	100	9	98.2	5	90.0	10	93.8	47	96.9	20	86.2

At the beginning of the fifth year interval (Table VIII), 90 per cent of the patients with far advanced disease who had strict bed rest three months or less were alive. One died of a nontuberculous pelvic operation; the other of reactivation and extension of pulmonary tuberculosis. At the beginning of the fifth year interval (Table VIII), 86.2 per cent of the patients with far advanced tuberculosis treated by strict bed rest more than three months following the institution of artificial pneumothorax were alive. One died following a pelvic operation for a nontuberculous condition; two died from reactivation and spread of the disease to the opposite lung after artificial pneumothorax had been discontinued; one died from miliary tuberculosis; and two others have died subsequent to this period of spontaneous pneumothorax of the contralateral lung.

Table IX shows the total number of months each group of patients has spent on strict bed rest before artificial pneumothorax was instituted, and immediately following the institution of collapse and subsequently.

TABLE IX

	STRICT BED REST THREE MONTHS OR LESS				STRICT BED REST MORE THAN THREE MONTHS			
	NO. CASES	BEFORE TREATMENT	IMMED. AFTER	SUBSEQUENTLY	NO. CASES	BEFORE TREATMENT	IMMED. AFTER	SUBSEQUENTLY
Min.	42	33	33½	20	16	35	211	54
M. A.	54	79	51¾	35	70	175	1,044	253
F. A.	23	57	16½	86	34	69	674	85

Among the forty-two minimal cases who had three months or less of strict bed rest at the time artificial pneumothorax was instituted, only one had previously been treated in an institution. This young man spent thirty-three months in a sanatorium. Soon after his discharge, he had a frank pulmonary hemorrhage. Artificial pneumothorax was immediately instituted, and he has required no other treatment. Of the entire forty-two patients with minimal disease, a total of thirty-three and one-half months was spent on strict bed rest during and immediately following the institution of artificial pneumothorax. Twenty-three of these patients had no bed rest, that is, they have been entirely ambulatory throughout the course of their treatment. Seven spent a total period of fourteen and one-fourth months in bed in their homes, ranging from one week to three months each. Twelve spent a total period of nineteen and one-fourth months in a general hospital, ranging from one week to three months each. One of these patients was treated in sanatoriums. All have been advised to lead well-regulated lives, and to avoid all exposure whatsoever to open cases of tuberculosis.

Three of the forty-two patients with minimal disease have subsequently spent a total of twenty months in institutions, two with no medical reason, but wholly on the advice of others who were convinced that institutional care is absolutely essential for the control of tuberculosis. Neither of these patients had tubercle bacilli in the sputum or any symptoms of tuberculosis. The third was advised by a physician to discontinue artificial pneumothorax after a period of eighteen months of treatment. Her disease later reactivated, and extrapleural thoracoplasty became necessary.

Of the sixteen patients with minimal disease who spent more than three months on strict bed rest immediately following the institution of artificial pneumothorax, a total of thirty-five months was spent on bed rest before treatment was begun. The four patients who devoted this time to bed rest had their treatment instituted while still in the institution, or while still on bed rest at home; in other words, it did not appear that strict bed rest alone would suffice to control their disease. Immediately after artificial pneumothorax was instituted and before becoming ambulatory, the sixteen patients spent 211 months on strict bed rest in homes or in hospitals. Five were strict bed patients over a total period of thirty-three months at home; eight devoted a total of 160 months to hospitals and sanatoriums, and three spent eighteen months in hospitals and strict bed rest at home. Two of the sixteen patients later returned to bed rest; one, approximately seven years after pneumothorax was instituted, developed mixed infection empyema through a pleuropulmonary fistula; the other patient's progress was excellent, and, for no medical reason whatsoever, he entered a sanatorium purely on the advice of nonmedical friends. These two patients have devoted fifty-four months to bed rest, after having previously been ambulatory.

Of the fifty-four patients with moderately advanced tuberculosis who were on strict bed rest three months or less at the time artificial pneumothorax was instituted, five had spent a total of seventy-nine months in institutions. One of these patients was in a sanatorium for twenty-eight months, during which time the phrenic nerve was interrupted, but no attempt was made to collapse her lung by artificial pneumothorax. Soon after discharge from the sanatorium her disease reactivated, and she had a rather profuse pulmonary hemorrhage. Artificial pneumothorax was immediately instituted, and she has since been ambulatory. Three patients were treated in the institution by strict bed rest alone. One had artificial pneumothorax instituted just before leaving the institution. Twenty-eight of the fifty-four patients had no bed rest when artificial pneumothorax was instituted. Fifteen spent a total of thirty-one months on strict bed rest at home; nine a total of sixteen and one-fourth months in hospitals, and two a total of four and one-half months in hospitals and homes. Thus, twenty-six of the fifty-four patients spent a total of fifty-one and three-fourths months on strict bed rest, during and immediately after artificial pneumothorax was instituted. Three of the fifty-four patients have subsequently spent thirty-five months on strict bed rest, one of whom entered an institution when a large pleural effusion developed; another after empyema developed, and thoracoplasty became necessary; the third after the pneumothorax was lost through the development of obliterative adhesions, and the disease reactivated.

Among the seventy patients with moderately advanced disease, who had more than three months of bed rest immediately following the institution of artificial pneumothorax, twelve devoted a total of 175 months to bed rest before artificial pneumothorax was instituted. Most of these patients entered institutions in the day when watchful waiting was thought to be indicated, that is, when it was believed that everyone should have a trial of strict bed rest to determine whether nature would control the disease before considering collapse therapy. One of these twelve patients spent five and one-half years in a sanatorium with unilateral disease, which slowly progressed on strict bed rest. After pneumothorax was instituted, she was ambulatory in a few months, and for the past seven years has carried on all the duties of her household. Immediately following the institution of artificial pneumothorax, thirteen of the seventy patients spent eighty-seven months on strict bed rest at home. Forty-seven spent 780 months in sanatoriums and hospitals. The remaining ten spent 177 months in hospitals and on strict bed rest at home. Thus, 1,044 months, or eighty-six years, were spent in inactivity immediately following the beginning of collapse therapy. Of these seventy patients who became ambulatory, twelve later returned to bed. Four lost their collapse, and the disease reactivated, one of whom died of tuberculosis at the age of seventy-five years; two are still in institutions, one already having spent thirty-nine months there; the fourth is now on full working capacity. Two patients voluntarily

discontinued pneumothorax, and their disease reactivated; one has died and the other has hopelessly advanced disease. Three, because of ineffective collapse and poor general health, have spent considerable time in institutions or on strict bed rest at home (one seventy-two months in a sanatorium), and are now apparently hopelessly advanced cases. One patient developed empyema and required thoracoplasty. Two have returned to bed rest for short periods of time on their own initiative. These twelve patients have spent a total of 253 months, or more than twenty-one years, in institutions or on strict bed rest in their homes.

Among the twenty-three patients with far advanced disease who spent three months or less on strict bed rest, five had been hospitalized for a total period of fifty-seven months before artificial pneumothorax was instituted. One had interruption of the phrenic nerve in the institution, but artificial pneumothorax was not attempted. Soon after discharge the disease reactivated and ambulatory artificial pneumothorax was instituted. One patient had spent twelve months in an institution with no attempt to collapse the lung. Her disease reactivated soon after discharge. Another spent ten months in a sanatorium on bed rest alone. After leaving the institution, a large cavity made its appearance in the upper lobe of the right lung. She refused to return to an institution, and artificial pneumothorax was instituted. Another had artificial pneumothorax instituted just before leaving the sanatorium. The fifth patient had been institutionalized for twenty-four months and left against advice. Artificial pneumothorax was then instituted purely as a compromise, mainly in an attempt to render his sputum negative, and prevent the spread of his disease to others as long as possible. Of the twenty-three patients, fourteen had no bed rest when artificial pneumothorax was instituted. Three spent a total of six and one-fourth months in bed at home; six spent a total of ten and one-fourth months in hospitals. Thus, the twenty-three patients spent a total of sixteen and one-half months in bed immediately following the institution of artificial pneumothorax. Subsequently five of these patients spent eighty-six months in institutions. One was advised to enter a sanatorium for intrapleural pneumonolysis. Another, whose artificial pneumothorax had been instituted as a compromise because he had previously been institutionalized and refused to return, developed mixed infection empyema through a pleuropulmonary fistula. Another had extension of disease to the opposite lung. The remaining two patients lost their collapse through the development of obliterative adhesions, and their disease reactivated.

Among the thirty-four patients with far advanced disease who spent more than three months on strict bed rest immediately following the institution of artificial pneumothorax, eight had devoted sixty-nine months to hospitalization before collapse therapy was instituted. Immediately after treatment was begun, six devoted thirty-nine months to

strict bed rest at home; twenty-six were hospitalized 616 months; and two devoted nineteen months to hospitalization and strict bed rest at home. Thus, a total of 674 months, or more than fifty-six years, of inactivity was spent by these patients. After having become ambulatory, five of the thirty-four patients returned to bed. Two lost their collapse through obliterative adhesions, one of whom died, and the other required thoracoplasty. One, because of unsatisfactory collapse, developed definite spread of the disease and died; one developed bloody effusions and required thoracoplasty; the fifth returned to the institution for only three months for general building up. This patient now has full working capacity.

In none of the 239 patients just reported did disease in the opposite lung become a serious complication while artificial pneumothorax was continued. Among those who were on strict bed rest three months or less, none of the forty-two minimal cases have developed demonstrable lesions in the contralateral lung. Of the fifty-four patients with moderately advanced disease, two developed small lesions which have subsided without causing illness. A third, after the collapse had been lost by obliterative adhesions and the disease reactivated, developed rapidly progressive tuberculosis in the opposite lung as a terminal event. Of the twenty-three far advanced cases, one discontinued pneumothorax, after which the disease reactivated, and near the time of death, extensive, rapidly progressive disease appeared in the opposite lung. Two developed empyema, and later disease appeared in the contralateral lung.

One of the sixteen minimal cases which had more than three months of strict bed rest developed empyema, and later significant disease appeared in the contralateral lung. Among the seventy patients with moderately advanced tuberculosis when treatment was begun, three voluntarily discontinued artificial pneumothorax after which the original disease reactivated and spread rapidly through the other lung. Two have died, and one is apparently in the terminal stage. Two patients lost their collapse through the development of obliterative adhesions. Following the reactivation of the original disease, lesions made their appearance in the opposite lung; one has died and the other is still in a sanatorium. Two patients whose pneumothorax became ineffective through the development of obliterative adhesions had reactivation of disease that spread to the opposite lung. Among the thirty-four patients with far advanced disease, two lost their collapse through adhesions, their disease reactivated and later spread to the opposite side before death.

In addition to the 239 patients in whom only unilateral disease could be demonstrated at the time treatment was begun, there are in Group I sixty patients who had bilateral disease when artificial pneumothorax was instituted, or developed significant lesions in the contralateral lung

while on treatment. Of those who had bilateral disease when artificial pneumothorax was instituted, twenty had strict bed rest three months or less. Only one of these patients had minimal disease. She was treated by ambulatory pneumothorax on one side and has had no complications or increase in disease. Thirteen had moderately advanced disease, four of whom had partial bilateral pneumothorax instituted. One had a phrenic nerve interrupted. This patient also required intrapleural pneumonolysis. Four have developed obliterative adhesions. Two of the thirteen later required strict bed rest; two have died of tuberculosis, one of miliary disease, and the other of reactivation following loss of collapse through obliterative adhesions. Another died of nontuberculous meningitis. Six of the twenty patients had far advanced disease when treatment was instituted. One had interruption of a phrenic nerve. Three stopped artificial pneumothorax treatment; two, because cavities could not be closed, and thoracoplasty was recommended. Both have entered institutions; the third upon recommendation with good recovery.

Nineteen patients who had bilateral disease when artificial pneumothorax was instituted were on strict bed rest more than three months. Only one had minimal disease. This patient later required bilateral artificial pneumothorax and at the present time has good working capacity. Thirteen had moderately advanced disease. Phrenic nerve interruption was performed for one; three required bilateral pneumothorax; six discontinued pneumothorax because of obliterative adhesions; five returned to institutions because of spread of disease, two of whom have again become ambulatory; two are still in the institution; and one has died. Five patients had far advanced disease when artificial pneumothorax was instituted. One developed tuberculous empyema, which was treated successfully by oleothorax. This patient has also had interruption of the phrenic nerve. Two of the five required bilateral artificial pneumothorax; one was hospitalized after having become ambulatory; two have died of tuberculosis, one of miliary disease and the other of spread of disease in the contralateral lung.

Nine patients who had unilateral disease when treatment was first instituted but later developed lesions in the contralateral lung, received three months or less of strict bed rest. Two of these patients had minimal disease when treatment was instituted, one of whom very soon developed extensive bilateral tuberculous cervical adenitis, tuberculous laryngitis, and rapidly progressive disease in the contralateral lung, and died of tuberculosis; the other, after developing a small lesion in the contralateral lung, was sent to a sanatorium, where bilateral pneumothorax was not found necessary, and the x-ray shadow has almost entirely disappeared. Seven patients had moderately advanced disease, one of whom had phrenic nerve interruption; one, thoracoplasty. Three of

the seven required bilateral pneumothorax. Four discontinued artificial pneumothorax because of adhesions; one voluntarily discontinued treatment, after which his disease reactivated and spread to the opposite lung. Four were later institutionalized, one because of pregnancy, who delivered a healthy infant and now has a full working capacity; one, because of the appearance of disease in the opposite lung and severe diabetes; one, because of reactivation and extension of tuberculosis following voluntary discontinuation of artificial pneumothorax; and the fourth, because of reactivation and extension of disease following loss of collapse through obliterative adhesions. This patient died. There were no far advanced cases in this group.

Among the patients who began with unilateral disease and later had extension to the opposite lung and were treated by bed rest for more than three months, there was none with minimal disease. Nine had moderately advanced disease, one of whom developed empyema. Bilateral artificial pneumothorax was employed in seven of the nine cases; three patients were compelled to stop artificial pneumothorax on one side, because of the development of adhesions. Five returned to strict bed rest, after having become ambulatory. Three patients who had more than three months of bed rest had far advanced disease when artificial pneumothorax was instituted; two of them have required bilateral pneumothorax, one of whom had treatment instituted on the left side in 1921. A fairly extensive area of disease made its appearance in the right lung early in 1936. This patient is responding well to ambulatory artificial pneumothorax. The other had coexisting diabetes and died of tuberculosis, approximately ten years after artificial pneumothorax was instituted.

Group II consists of thirty-five patients who have been treated by artificial pneumothorax and strict bed rest who never became ambulatory while on artificial pneumothorax treatment. Two of these patients had only minimal unilateral disease when they were first hospitalized and artificial pneumothorax instituted. One of them lost her collapse through the development of obliterative adhesions, and thoracoplasty was later performed. She had been in bed for approximately nine years and now is suffering from coexisting malignancy. The other patient with minimal disease, although strict bed rest and artificial pneumothorax were employed, developed extension of the pulmonary disease. Later tuberculous peritonitis was diagnosed from symptoms and laparotomy. This patient has been institutionalized approximately five years. Ten of the thirty-five patients had moderately advanced disease when treatment was begun. They have devoted long periods of time to strict bed rest, mostly in institutions. Five have died, one of whom had spent six and one-half years in sanatoriums. Three have required thoracoplasty, two of whom are now ambulatory, and one is still on

strict bed rest; one became ambulatory after artificial pneumothorax was discontinued. The tenth patient lost her collapse through the formation of obliterative adhesions; later the phrenic nerve was interrupted and thoracoplasty recommended. She has refused chest surgery and remains in a sanatorium, after having spent approximately nine years in such institutions.

Twenty-three of the thirty-five patients had far advanced disease when artificial pneumothorax was instituted. After long periods of time, sixteen have died, all of tuberculosis except two, one of whom died of malignancy and the other of nontuberculous pneumonia. Two of the twenty-three have had successful thoracoplasties. Both are working on a full-time basis. Three now have bilateral, far advanced disease which is apparently hopeless. One of the twenty-three had artificial pneumothorax discontinued, because it was impossible to close a cavity; the phrenic nerve was interrupted, and this patient is now semiambulatory. One patient had disease appear in the contralateral lung, has had bilateral pneumothorax instituted, and remains in the sanatorium, where she has been for more than four years.

Group III consists of more than one hundred patients for each of whom we have administered only a few treatments. In this group are some transients whose work or vocation brought them into this community temporarily. A good many of the patients in this group were given only the first few treatments for other physicians, who desired to do the subsequent work. Most of these physicians had not previously employed artificial pneumothorax among their private patients and desired assistance in beginning the treatment on the first few patients. Of all of our artificial pneumothorax work, this has been the most gratifying, for it has helped to equip young physicians to carry on with this valuable therapeutic and public health measure among their tuberculous patients.

Group IV consists of ninety-two patients for whom artificial pneumothorax was recommended, but it was impossible to find free pleural space to contain more than small pockets of air which did not prove adequate to control the disease, and in the majority of these cases, no free pleural space could be located. In twenty-two of them, because of bilateral disease, the patient's general condition, etc., surgical collapse was not indicated. All of these patients went on until death, some of them after many years of hospitalization.

Fourteen of the ninety-two patients in this group had interruption of the phrenic nerve, four of whom have died of tuberculosis. In three of these patients, thoracoplasty was not indicated because of extent of disease. The fourth, however, had thoracoplasty strongly recommended because the disease was chronic and unilateral, but for religious reasons refused. Three more of the fourteen who had interruption of the phrenic nerve have since had reactivation of their disease with

tubercle bacilli in the sputum. Thoracoplasty is not indicated because of extent of disease. Six of the fourteen patients have good working capacities. One of the fourteen has not been traced.

Of the ninety-two patients, twenty-one have had extrapleural thoracoplasty, four of whom have died; two of tuberculosis and two of acute pneumonia. Seven of the twenty-one patients had thoracoplasty performed during the past five years, all of whom are doing well. In the remaining eleven, the thoracoplasties were performed from five to twelve years ago, and all have good working capacities.

Of the ninety-two patients in this group, thirty-four have not had surgical collapse. Eight of them are now in sanatoriums, six have advanced disease but refuse institutional care. Three of the thirty-four have had institutional treatment but have been discharged, one after a period of approximately seven years in the sanatorium. Nine of the thirty-four patients have had long periods of bed rest in their homes, all of whom are now ambulatory and eight are working. One has refused bed rest and is still working. Seven of the thirty-four patients have not recently been traced.

DISCUSSION

In 1933, attention was called to the fact that our work on strictly ambulatory artificial pneumothorax began in 1926 with a patient who supposedly was on strict bed rest in the home after artificial pneumothorax was instituted. It was later learned that she never discontinued work. Although she had small cavities present, tubercle bacilli in the sputum, and definite symptoms present, she responded as well to pneumothorax alone as any patient who had ever been observed to respond on this treatment, plus strict bed rest. Her sputum quickly disappeared and in a short time she was in good general health. At the end of three years, artificial pneumothorax was discontinued, and she has remained in good health with full working capacity. Immediately following this demonstration of effectiveness of artificial pneumothorax alone, this method of treatment was recommended to a number of patients with minimal and moderately advanced disease. The results obtained were so satisfactory that at the present time it is frequently recommended. It is often preferable, however, to have the patient on strict bed rest while the first few treatments are being administered. When this cannot be arranged, satisfactory results may be obtained without strict bed rest.

Among the patients treated by artificial pneumothorax with three months or less of strict bed rest, there are several with advanced disease for whom such treatment was not originally recommended. They were advised to be institutionalized; first, because they were already known to have positive sputum or were potential spreaders of tubercle bacilli; and second, because of the extent of their disease. However, some of

them were in good general health and had always worked on a full-time basis. The fact has long since been established that a considerable number of persons with frank pulmonary tuberculosis in a chronic form are never incapacitated enough to give up work; they may live active lives into the seventies and eighties. I have recently seen a man who died at the age of eighty-two years and was very active until near the time of death. It is true that he gave a history of cough and rather profuse expectoration which had persisted over the past thirty years. These symptoms were attributed to bronchitis; they never reduced his working capacity, and he never considered them sufficiently significant to consult a physician. It was not until two of his daughters, one forty-five and the other fifty-six years, fell ill from tuberculosis and the family physician sought the source that this man's disease was brought to light. Thick-walled cavities were present in both upper lobes, and tubercle bacilli were abundantly present in his sputum. When this type of tuberculosis is detected in earlier life, the patient may be so free from symptoms that he is likely to refuse hospitalization no matter how firmly convinced the physician is of its necessity. In most parts of the country, there is no recourse to ordinances or laws that will compel hospitalization. With such patients the physician cannot wash his hands of the case, but will do everything possible to control the disease by other methods, always believing that if his treatment fails and hospitalization continues to be indicated, he will ultimately secure the confidence of the patient so that his recommendations will be accepted.

Compromises are frequently necessary in every phase of tuberculosis work. Even among the patients who are institutionalized, compromises are often made but sometimes fail. In writing of patients discharged from the Minnesota Sanatoriums in 1935, Hilleboe[34] says: "Perhaps the greatest handicap the tuberculosis sanatoria labor under is the inability to keep patients in residence until maximum care has been administered. Of the 1,200 patients discharged with adult type tuberculosis, 238 (20 per cent) left against advice or for disciplinary reasons . . . . ; 94 (40 per cent) of these cases had positive sputum." In several of our compromise patients we have succeeded in collapsing the lung by artificial pneumothorax so as to render them free from sputum or negative for tubercle bacilli. They have continued their work as safe associates to others. In a few, we have failed to collapse cavities but have been able finally to convince the patients that other forms of collapse therapy, particularly thoracoplasty, were necessary. In the occasional case, we have failed utterly either to close cavities or convince the patient of the necessity for thoracoplasty or hospitalization for his own sake, as well as that of his family and community.

A few of our failures have been due to the fact that we persisted too long in attempting to collapse the lung when adhesions prevented the

closure of cavities. When closure cannot be accomplished within a reasonable period of time, artificial pneumothorax should be discontinued, no matter how much the patient insists upon further attempts. If it is not discontinued, whether the patient be on ambulatory treatment or strict bed rest, there is considerable likelihood of the contents of the cavity finding its way into the pneumothorax space and setting up mixed infection empyema. A typical case is that of a young man who had artificial pneumothorax instituted on March 16, 1932. His disease had existed for many years. A large cavity was present in the left upper lobe. The remainder of the left lung, which also contained considerable disease, was well collapsed, but the cavity remained open. Having been previously treated in a sanatorium, he refused to be hospitalized. Although chest surgery was strongly recommended many times, he decided to postpone any such procedure and later developed mixed infection empyema. He has since been a strict bed patient and, because of the empyema, the thoracoplasty has been postponed indefinitely.

To continue artificial pneumothorax after it has been definitely determined that cavities cannot be closed by this method may be worse than no treatment, since the sputum usually remains positive for tubercle bacilli. Moreover, a patient may be given false sense of security and open cavities are potent sources of hemorrhage and mixed infection empyema. It is far better, therefore, to discontinue pneumothorax and recommend other collapse procedures, such as extrapleural thoracoplasty, provided they are not contraindicated by such conditions as too extensive disease in the opposite lung.

There has been considerable controversy over the treatment of tuberculous patients in the home. So far as arrest or recovery from the disease is concerned, Pratt[35] obtained as good results in the homes as others obtained in institutions. After all, the chief reason for sending any patient to the sanatorium is to prevent the spread of his disease to the members of his family and other associates. Indeed, some of our sanatorium medical directors have already adopted the policy of discharging patients very quickly after admission if they are found to have negative or no sputum, and to discharge others who have had positive sputum in a very short time after it has become negative. All of these patients are kept under the close observation of their private physicians or the sanatorium staff. This is definitely a step forward for it makes available many beds for communicable cases of tuberculosis, which in the past were occupied over long periods of time by patients whose communities were not benefited by their isolation. With our modern diagnostic procedures, which when properly applied, uncover developing pulmonary lesions in the presymptom stage before tubercle bacilli are present in the sputum, as well as some more extensive lesions causing mild symptoms which the patient attributes to other causes, and the

sputum which he believes comes only from his throat and is due to a catarrhal condition, yet which frequently contains tubercle bacilli, we are able to institute treatment, particularly artificial pneumothorax, while it is capable of preventing the sputum from ever becoming positive in some cases, and quickly rendering the sputum negative in others. The majority of such patients can be treated adequately in the home and many of them need not discontinue their work for more than a few weeks if at all. The source of their disease, however, should be sought and contact broken with any and every open case of tuberculosis, for continued reinfection with tubercle bacilli may result in the appearance of new lesions in their bodies or definite retardation of the control of their disease.

The argument is frequently advanced that the complications of artificial pneumothorax, such as empyema, occur with such frequency as to contraindicate this treatment in minimal cases. In reality, none of our forty-two patients with minimal disease treated on an ambulatory basis has developed empyema, and only one of the sixteen patients treated by artificial pneumothorax and more than three months of strict bed rest has developed this condition. Indeed, it has also been very rare among the moderately advanced cases, only 5.5 per cent of the ambulatory and 4.2 per cent of the patients who had long periods of strict bed rest. Among the minimal patients of both groups there has not been a single case of embolism, accidental or spontaneous pneumothorax. Space does not permit the inclusion of a discussion of conversion of sputum or serous pleural effusions. These subjects will be discussed in another article.

In times past approximately 80 per cent of the persons with pulmonary tuberculosis had their disease detected after it was well advanced and when they were definitely ill, so that emaciation, high fever, rapid pulse, and other symptoms definitely indicated bed rest. The majority of them were communicable cases. With disease so advanced, there was little to do except to have them lie in bed, and trust that they had enough resistance to control it. Bed rest was advised over long periods of time, and it was truly indicated for such patients and still is. Hilleboe,[36] however, has recently called attention to the fact that among patients discharged from sanatoriums, those with far advanced disease have the risk of dying of tuberculosis increased forty times (over that of persons in the general population from which the patients were drawn); those with moderately advanced disease, sixteen times; and even those with minimal disease, four times.

Long periods of hospitalization are always bad for the morale of the patient, but when one suffers from a disease that is communicable and remains so even with the best known treatment, it is a matter of choosing between two evils; the one, to allow the patient to remain in the com-

munity and spread his tubercle bacilli everywhere; the other, to hospitalize him with full knowledge of the consequences from the standpoint of loss of morale, development of nervous symptoms, etc. Most certainly from the standpoint of the community, one must choose the former. Long periods of hospitalization create a serious rehabilitation problem. There is no better evidence of this fact than the present development of rehabilitation programs, which are rapidly becoming an important phase of tuberculosis work. I have seen among patients who were previously stable, a number of nervous symptoms develop during long periods of hospitalization. In some of them even though their disease is controlled so that they are physically able to work, they will never resume their previous occupations or undertake new work. Their attitude toward life has become so changed that they are misfits in their homes and in their communities. In writing of patients discharged from sanatoriums Stearns[37] says: "Tragically this is a psychopathic condition which sometimes persists. Discharged as arrested cases with no active tuberculosis, certain patients fail to regain a normal perspective and always see themselves and their disability as the center around which all else revolves. Discharged as well, they never recover." A serious attempt is being made in many of our institutions to prevent such situations among patients who must be institutionalized through the development of vocational training.

The cost of hospitalization of tuberculous patients constitutes a serious economic problem which can now be solved in part by ambulatory artificial pneumothorax. For example, of the fifty-four patients with moderately advanced disease who were treated with three months or less of strict bed rest, one finds an average of 0.95 months for each patient, or a total of 4.3 years. At a cost of $1,000 a year, this amounts to $4,300. The seventy patients with moderately advanced disease, treated with more than three months of strict bed rest, spent on an average 14.9 months each, immediately after artificial pneumothorax was instituted, making a total of eighty-seven years. At a cost of $1,000 per year this amounts to $87,000. If the seventy patients had been treated on the ambulatory basis, like the fifty-four, the total period of bed rest would have been 5.54 years at a cost of $5,540. Thus, a saving of $81,460 might have been effected. On the other hand, if the fifty-four patients who were treated by the ambulatory method had been treated by the other method, the total period of bed rest would have been sixty-seven years at a cost of $67,000. Since their cost was actually $4,300, a saving of $62,700 was effected. One might argue that the fifty-four patients would subsequently require much more hospitalization than those who had long periods of bed rest immediately following artificial pneumothorax, but actually they have spent a total of only thirty-five months in bed, whereas the seventy patients treated originally with a longer period of bed rest have subsequently spent a total of 253 months in bed.

These figures could be extended throughout the other groups reported with an ever widening disparity between actual cost of ambulatory pneumothorax and institutional treatment. However, the point is sufficiently clear, save that the cost of ambulatory pneumothorax is within the range of the ordinary individual's income, while the cost of prolonged bed treatment is not. The actual saving in dollars and cents, however, is insignificant in comparison with the loss of the patient's time, the mental and nervous strain for the patient and his family, as well as the rehabilitation problem which so frequently develops following long periods of institutionalization.

Since ambulatory pneumothorax in properly selected cases fulfills the three main objectives in the treatment of pulmonary tuberculosis: checking the spread of disease, conversion of positive to negative sputum, and restoration of the patient's working capacity in the shortest time possible; since the complications arising from such a procedure are no greater than strict bed rest and pneumothorax, this must naturally be the method of choice for those patients in whom it is possible.

SUMMARY

1. A brief review of the literature on collapse therapy in the minimal stage of pulmonary tuberculosis and ambulatory artificial pneumothorax treatment in various stages of the disease indicates that this type of treatment of tuberculosis is being extended.

2. One hundred and nineteen patients with unilateral disease, treated by ambulatory artificial pneumothorax, are compared with one hundred and twenty patients with unilateral disease, treated by pneumothorax and strict bed rest for periods exceeding three months.

3. From the standpoint of development of complications, there is no significant difference between the two groups among those with minimal and moderately advanced disease. Among the far advanced patients, 5.5 per cent more developed empyema in the ambulatory group than in the group treated by artificial pneumothorax and strict bed rest. This difference is equalized if the patients who refused strict bed rest and in whom artificial pneumothorax was undertaken on a compromise basis are excluded.

4. Fewer of the minimal and moderately advanced patients treated on the ambulatory basis later stopped work than of the minimal and moderately advanced cases treated with long periods of bed rest. Among the patients with far advanced disease, however, more who were treated by the ambulatory method stopped work. The percentage is higher because of the compromise cases; that is, those who refused the recommended procedures and in whom ambulatory artificial pneumothorax was instituted rather than allowing their disease to progress without any type of treatment are included.

5. Deaths from tuberculosis were considerably fewer in the ambulatory group than in the patients treated with strict bed rest in addition to artificial pneumothorax. However, the majority of the latter group have been followed for a longer period of time.

6. The chief reason for discontinuing artificial pneumothorax was the development of obliterative adhesions.

7. Attention is called to the contrast between the cost of treating patients by the ambulatory method and the strict bed rest method.

8. Sixty patients are reported who had bilateral disease when artificial pneumothorax was instituted or developed significant lesions in the contralateral lung while on treatment.

9. A group of thirty-five patients treated by artificial pneumothorax, but who have never become ambulatory, is reported.

10. A group of more than one hundred patients, including transients and those for whom artificial pneumothorax was only instituted for other physicians, is presented.

11. A group of ninety-two patients for whom artificial pneumothorax was found impossible because of adhesions is reported, showing the results of surgical procedures when administered.

CONCLUSIONS

1. Modern methods of diagnosis have made it possible to detect progressive, chronic, pulmonary tuberculosis in the presymptom stage, when the patient is in good general health and often before tubercle bacilli are being disseminated to others. Effort should be put forth to have these methods applied in every community, in order to have treatment instituted when it is of most avail.

The three essentials in the treatment of pulmonary tuberculosis: checking the spread of disease; conversion of positive to negative sputum; and restoration of the patient's working capacity in the shortest possible time, are embraced in ambulatory artificial pneumothorax, provided it can be successfully administered.

2. When the disease is found in the minimal stage and is known to be progressive, artificial pneumothorax should be instituted at once. Bed rest alone permits the progression of the disease in far too many patients to justify its exclusive use. All patients with unilateral progressive disease in the moderately or far advanced stage should have artificial pneumothorax attempted, provided there is no special contraindication.

3. In most patients with minimal unilateral pulmonary tuberculosis, many with moderately advanced unilateral disease, and some with far advanced unilateral disease, artificial pneumothorax may be safely undertaken on the ambulatory basis; that is, ranging from no period of strict bed rest up to a period of three months.

4. Even when bilateral disease exists, great benefit may be derived by carefully administering artificial pneumothorax on the side of the more extensive or progressive lesion. In some cases, partial bilateral pneumothorax is helpful. When a lesion makes its appearance in the contralateral lung which was previously clear and shows evidence of progressiveness, treatment may be discontinued on the side of the original disease, provided it is well controlled; otherwise, bilateral artificial pneumothorax may be indicated.

5. No patient whose cavities are not adequately closed after artificial pneumothorax has been given an adequate trial should be continued on this treatment, because of the dangers of spreading the disease to the opposite lung, the hazards of hemorrhage and empyema. The treatment should be discontinued and surgery recommended.

6. When artificial pneumothorax cannot be instituted because of adhesions, one may resort to surgical procedures, such as interruption of the phrenic nerve, and particularly extrapleural thoracoplasty, with excellent results in many cases.

7. Every patient who has the diseased lung collapsed by artificial pneumothorax, whether on ambulatory or the strict bed rest method, in the home or the institution, should be protected against all exposure to patients with positive sputum, in order to prevent reinfection from exogenous sources.

8. Since clinically the results of ambulatory pneumothorax are equal, and since the patient's status quo is maintained, thus avoiding long periods of inactivity, and the hazards of institutionalization, and since a significant monetary saving of the resources of both the patient and the community is effected, the use of ambulatory artificial pneumothorax in properly selected cases should be extended.

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STUDIES ON TUBERCULIN HYPERSENSITIVENESS

I. The Relation of Hypersensitiveness to Tuberculin to the Postthoracoplasty Reaction*

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THE development of our knowledge as it deals with the treatment of certain cases of pulmonary tuberculosis by thoracoplasty has done much to lower the mortality rates following such procedures. The present tendency to perform this operation in multiple stages and a better judgment of the tuberculous patient's ability to withstand such procedures have been prime factors in this decrease. It has been learned that a multiple-stage operation is preferable to the old procedure of removing segments of numerous ribs at one time. The patient with the older, more fibrous lesion has long been looked upon as a better operative risk than the one with a more recent, exudative type of disease. Improved operative technique and methods of anesthesia have also played an important part in making thoracoplasty an operation of low morbidity and relative safety. While these things are true, the postthoracoplasty reaction and postthoracoplasty death still exist, and little more is known today of their nature than was known when such operative procedures were new. The question of why the thoracoplasty patient dies has been the subject of much generalization. It has been the purpose of this study to gain added information on one of the more important sides of this subject.

When the literature which has accumulated over the past twenty years is reviewed and the causes of death noted, they are found to range from the rather general classification of septic state or toxemia to the still more vague and general postoperative cause of death—cardiac failure. While it is not denied that the cardiac musculature may, in numerous instances, have suffered grievous damage, such as loss of tone from long periods of bed rest, toxic myocardial damage during the exudative stage of the disease or the deleterious effects which may follow changes in the heart axis due to pulmonary fibrosis, it is felt that to pass off rather easily the cause of these deaths as being due to cardiac failure is far from telling the whole story.

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It becomes apparent by studying a series of postthoracoplasty fatalities that there is a similarity of symptoms common to approximately 50 per cent of the group. The immediate postoperative condition of the patient is good, but toward the evening of the first day or the morning of the second, the temperature begins to rise, the pulse rate has become elevated, and while the respirations may be normal, there may be at this time slight cyanosis. The lungs, on physical examination and as visualized on x-ray films, are, as a rule, clear. In the larger group of patients, the reaction ends at this point, the temperature falls slowly or sharply to normal, and the cardiorespiratory function is restored. In the fatal cases, the temperature continues to rise, the pulse becomes more rapid, cyanosis deepens, and dyspnea becomes marked. The lungs, by this time, may contain many râles and rhonchi. Death occurs, as a rule, in from 48 to 96 hours postoperatively, although it may vary in either direction from this mean.

The findings at autopsy have in the large been of little help in giving an adequate cause of death. The right heart may be dilated and contain questionable antemortem thrombi, but such findings in a patient whose pulse has become elevated to 180 beats a minute or higher, and whose oxygen want may have been extreme for hours cannot be interpreted as significant. The lungs may and often do show marked edema which could adequately explain both the dyspnea and the cyanosis. The edema, however, is a late, not an early, finding, and cyanosis may be marked and the lungs clear. Microscopic examination of the tissues obtained in the majority of cases shows little that has not already been discovered during gross examination.

A more careful consideration of the clinical picture will reveal that it comes far from simulating that of cardiac failure. The antecedent temperature elevation, at times, hours before other signs have appeared, the early cyanosis when the heart action is good and the lungs clear, weigh strongly against the thesis of cardiac failure.

As long ago as 1922, Thjotta[1] suggested that such reactions were due to autotuberculinization. Since that time, other investigators, apparently independently, have suggested autotuberculinization as the probable cause of death (Brunner 1924;[2] O'Brien 1933[3]). Recently, Graham, Singer, and Ballon[4] have devoted some space to a discussion of this phenomenon in their textbook.

It was reasoned by the above mentioned authors that tuberculin from the diseased lung is squeezed into the blood and lymph channels by the falling of the decostalized chest wall. Coryllos[7] in a recent article has taken exception to such a premise, stating that the lymph and blood flow from a collapsed lung is "slowed down or even suppressed."

McCordock and Ballon[6] in 1932 showed that, in some cases, the postthoracoplasty death was due to a spread of the cavity débris into the contralateral lung, with a resultant tuberculous pneumonia. That this

can happen is well known, and that the protein products of the tubercle bacilli are as capable of producing a pneumonia as the organisms themselves has also been shown. The type of death as described by McCordock and Ballon, however, does not cover those cases which die with signs of severe intoxication without signs of serious disorder in the good lung.

As the situation now stands, a cause of the postthoracoplasty reaction has been suggested, but no proof as to its true nature has been offered. It is known that the majority of tuberculous individuals are extremely sensitive to tuberculin and that in the tuberculous guinea pig, death may occur in from 8 to 24 hours after the intraperitoneal injection of as little as 0.1 c.c. of old tuberculin. There are also recorded cases of rather severe reactions in hypersensitive human beings who have either inhaled tuberculin or by error have received too large an injection of it.

It was reasoned, therefore, that the postthoracoplasty reaction might represent the union of antigen and antibody. If such union occurred, desensitization of the host would be expected, and the tuberculin skin test following thoracoplasty should become negative or of decreased positivity, depending on the intensity of the reaction. It was to prove, or disprove, this premise that the following work has been done.

METHODS

All skin testing was done with the intracutaneous test (Mantoux) method. Old tuberculin was used throughout in a dilution of 1:1000, and the test dose was 0.1 c.c. The dilutions were prepared fresh each week and stored in the ice box. The test material used in the work done at the Barnes Hospital was all from the same original stock of full strength old tuberculin. Likewise, the test solutions used for testing in that part of the work done at the Herman Kiefer Hospital were from the same original stock solution, although different from that used at Barnes Hospital.

Patients ready for thoracoplasty were skin tested from four days to one week before going to the operating room. They were again retested postoperatively when the temperature had reached, or approximately reached, normal. This varied in individual cases, but the mean was from the fourth to the seventh postoperative day. The testing was repeated from two to four days before the second-stage operation and again following the second stage in the same manner and at approximately the same time as following the original stage. If three stages of thoracoplasty were done, testing was done in the same manner as before and after the previous stages.

The skin tests were read at 24 and 48 hours. The degree of the reaction was based on the amount of edema present. The area of induration

was read in millimeters of induration, and ulceration or necrosis was noted. No attempt was made to grade on the basis of plus reactions. The measurements alone were taken as an indication of the degree of the reaction.

In the final study of results, the waning of cutaneous sensitivity postoperatively has been compared with the postoperative constitutional reaction, and an attempt has been made to correlate the state of the disease as evidenced by the x-ray films with the degree of the postoperative reaction.

RESULTS

This study has been based on 46 cases of pulmonary tuberculosis which were submitted to thoracoplasty.

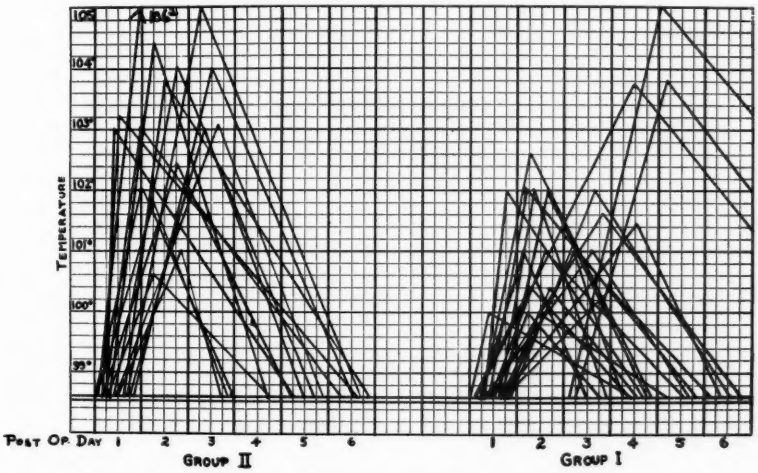

Chart I.—A comparision of the temperature reaction following first-stage thoracoplasty in Group I and Group II patients. Note the higher temperature elevation in Group II patients, and that in those cases in Group I where the temperature elevations were high, the day of onset was the second to third day.

It will be seen from examining the accompanying charts and tables that the cases may roughly be divided into two groups, namely, those which clinically reacted mildly following thoracoplasty, and those in which the postoperative clinical reaction was more vigorous. For purposes of reference, the former will henceforth be known as Group I, and the latter as Group II. There are 24 cases in Group I, and 22 cases in Group II.

The average postoperative temperature in Group I was 101, while that of Group II was 103.3 (Chart I) following the first stage. As will be seen by referring to Charts II and III, Group I cases evidenced little change in the skin sensitivity following thoracoplasty, while in Group

II cases, the decrease in skin sensitivity was marked. In those cases in Group II there was a fall in skin sensitivity which averaged 22 mm. of edema area as compared with a decline of 7 mm. in the case of the less sensitive individuals, at an average time interval of 5 days postthoracoplasty. It may be further noted that the second preoperative test, made on the average four days before the second-stage operation, showed a return of skin sensitiveness of 16 mm. of skin edema area in the case of Group II patients, as compared with no change between the two tests in Group I patients. Following the second-stage operation, there was a decrease in skin sensitivity of 19 mm. of induration in the case of Group

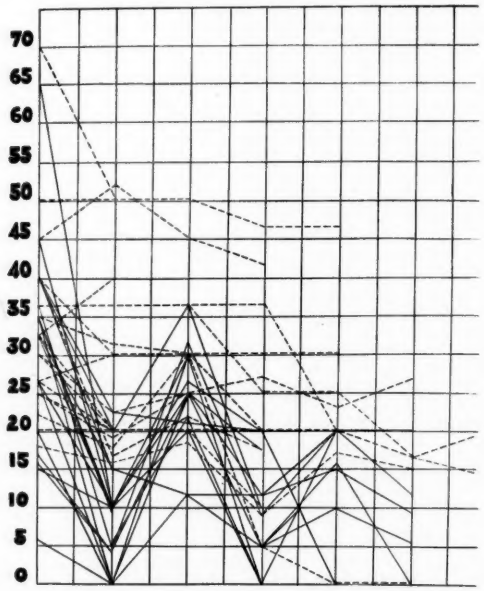

Chart II.—Broken lines represent Group I. Unbroken lines represent Group II. Note the general absence of sharp declines in skin sensitivity in Group I as compared with Group II.

II patients as compared with a decrease of 3 mm. in the case of Group I. Skin sensitiveness returned in the Group II patients 10 mm. during the period between the second- and third-stage operations, while in Group I there was a continued fall of 3 mm. The skin test following the third stage showed a fall of 6 mm. in Group II, while in Group I there was no change in the skin test.

There was in both groups a general tendency to lose skin sensitivity as is evidenced by a mean loss of 20 mm. of induration by Group II, and 15 mm. by Group I between the first preoperative test and the last postoperative test (Chart III).

The general tendency of the Group II patients to react violently at times when compared with Group I is more graphically indicated in Chart II. Here it may be seen that following each stage the reacting group showed sharp declines in skin sensitivity, while the nonreactors are in the main represented by lines more nearly horizontal.

In view of the fact that there were apparently marked differences in cases of the same apparent skin sensitiveness in their manner of post-thoracoplasty behavior, an attempt was made to correlate the type of lesion with the patient's postoperative stability or instability. In Group I or those cases showing little postoperative reaction, ten cases were of the productive type, four were empyemas, and in two cases, the thoracoplasty was done over an existing pneumothorax which had been present for some time. In two instances the lesion was of the mixed type, and

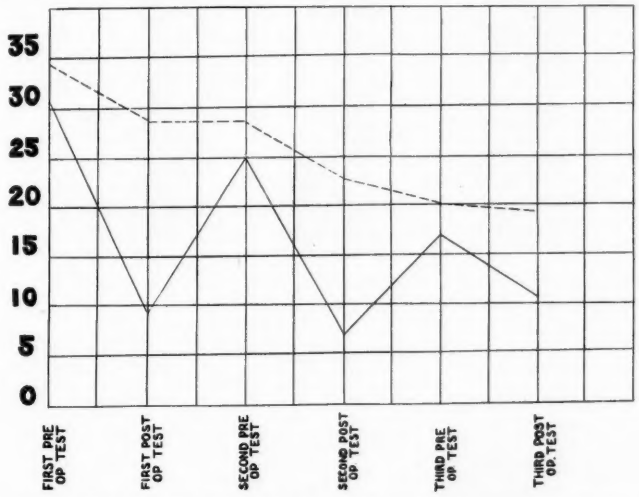

Chart III.—The abscissa represents the area of induration following 0.1 c.c. of 1:1000 O. T. Broken line shows the gradual loss of cutaneous hypersensitiveness in Group I patients. Unbroken line shows marked loss of sensitivity in Group II patients with return of sensitivity before next stage.

twice it consisted of a large apical cavity with little perifocal reaction. In the productive lesions in this group, a large area of pulmonary infiltration was not the rule, and the lesions in the main consisted of large cavities with surrounding fibrosis. In contrast to this is the type of case seen in Group II. Here all the cases, with the exception of three, were of the exudative or mixed types. There were large areas of consolidation throughout a greater or lesser part of the lung. In four instances, the x-ray had the appearance of the "good chronic," that is, large cavities with evidence of atelectasis. A review of the history in these cases revealed that they were all recent lesions, one having had a clear lung seven months before. In another case, although the history

of disease dated back three years, there was a history of recent exacerbations. From this angle of the study, there appeared to be some correlation between the age of the disease and the severity of the postoperative reaction. It also appeared that those cases in whom the amount of parenchymal infiltration was marked, although old, reacted more vio-

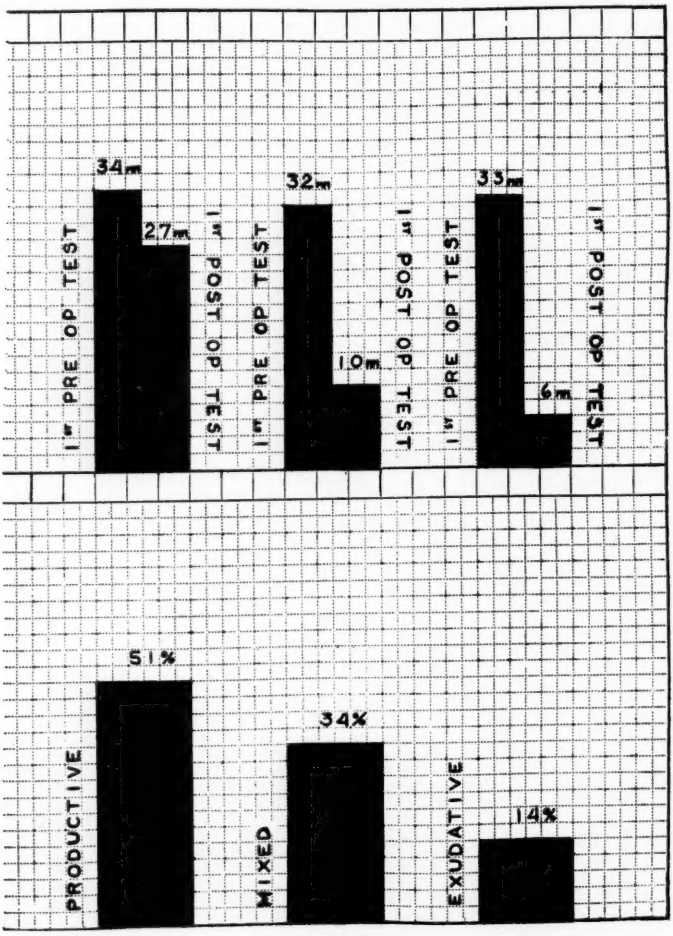

Chart IV.—A comparison of the loss of cutaneous sensitivity in productive, mixed, and exudative types of lesion. Note the marked loss in the last two, while there is little change in the former. The classification was not based purely on x-ray findings, but on a combination of x-ray findings, clinical course, and history.

lently and became desensitized more completely than other cases of similar age but with less pulmonary infiltration. The correctness of this premise is seen by referring to Chart IV. Here the degree of desensitization observed postoperatively is compared with the original

cutaneous sensitivity. It will be noted that 51 per cent of the cases were of the productive types and that the loss of skin sensitization in the group was slight. This reaction may be compared with marked loss of skin sensitization in the mixed and exudative groups following thoracoplasty.

DISCUSSION

These observations furnish support for the views previously advanced, namely, that the reaction following thoracoplasty is basically one representing a rather violent union of antigen with antibody, as a result of expressing tuberculin from tuberculous lung tissue at the time of thoracoplasty.

From the results here recorded, it is apparent that the reaction observed in certain cases of thoracoplasty following operation is in some way associated with that patient's tuberculin hypersensitiveness. The general rule which may be applied from the results would be that a soft lesion associated with a high degree of tuberculin sensitiveness affords the surgeon a poorer operative risk than one whose sensitiveness to tuberculin is lower, or whose lesion is older. Throughout this series, it was almost invariably the case that a patient with a high degree of sensitiveness to tuberculin and a relatively exudative or mixed lesion had the most marked postoperative reaction. It was also this type of case which showed the largest degree of postoperative desensitization (Chart IV). Such cases may be compared with those which, while exhibiting a high degree of skin sensitiveness, were the possessors of a productive lesion with small cavities on one hand, or with those whose lesions were composed almost wholly of cavity with little parenchymal involvement (Cases 2 and 7). In such individuals, there was little postoperative reaction, and in the majority of instances, no marked change in the skin sensitiveness.

Long and Wells[5] have extracted tuberculin from the tuberculous lung. While no record exists of experimentation which might show the comparative amounts of tuberculin in tuberculous lungs in the exudative and productive stages of the disease, it seems reasonable to venture the assumption that the amount would be higher in the exudative stage than in the older and more productive stages, for in the latter, much of the soft, caseous material which contains the tubercle bacilli and its products has been coughed up.

The rather sudden fall of the chest wall upon such lesions must of necessity squeeze varying amounts of tuberculin from the lung into the lymphatics and general circulation. The reaction of the patient incident to the expulsion of tuberculin into the general circulation will naturally vary directly with the sensitiveness of the individual and the quantity of tuberculin expressed. Thus the reaction manifested by the cases cited above is but natural in view of the type of lesion and the

TABLE I

RÉSUMÉ OF CASES

NO.	TYPE OF LESION	CUT. SENSITIVITY	POSTOP. REACTION	DESENSITIZATION	REMARKS
1.	Tuberculous empyema	22 mm.	none	none	
2.	Five cm. apical cavity	50 mm.	mild	-10	
3.	Four cm. apical cavity	30 mm.	none	none	
4.	Tuberculous empyema	25 mm.	moderate	none	
5.	Apical cavities	30 mm.	moderate	none	
6.	Five cm. apical cavity	50 mm.	mild	none	
7.	Productive pneumothorax unsatisfactory	18 mm.	mild	none	
8.	Productive lesion of long standing	45 mm.	mild	none	
9.	Mixed lesion predominately productive	25 mm.	none	none	
10.	Large apical cavity pneumothorax	20 mm.	moderate	none	Retained secretions in bronchus
11.	Productive thick-walled cavity	32 mm.	mild	-10	Sauerbruch type
12.	Empyema—apical cavity	35 mm.	moderate	none	
13.	Scattered productive	35 mm.	mild	none	Between 1st and 3rd stage complete loss of sensitivity
14.	Previous thoracoplasty	25 mm.	none	none	
15.	Empyema	35 mm.	Temp. 103°. Infected wound	+10	
16.	Empyema	25 mm.	none	none	
17.	Numerous cavities productive	30 mm.	mild	-10	
18.	Productive unsatisfactory pneumothorax	35 mm.	moderate	none	
19.	Pneumothorax-fixed lung	40 mm.	moderate	-15	
20.	Empyema	30 mm.	marked	none	Wound infection
21.	Productive lost pneumothorax space	15 mm.	moderate	none	Retained secretions
22.	Productive apical cavity	35 mm.	moderate	-25	
23.	Apical cavity—mixed	37 mm.	moderate	-32	
24.	Mixed lesion apical cavity	20 mm.	marked	-20	
25.	Apical cavity	35 mm.	marked	-35	
26.	Mixed lesion above empyema base	20 mm.	marked	-20	
27.	Old lesion with exacerbation	35 mm.	very marked	-25	
28.	Apical cavity—empyema at base	18 mm.	mild	-10	
29.	Exudative apical lesion with cavity	30 mm.	marked	-30	
30.	Exudative lesion apical cavity	35 mm.	very marked	-25	
31.	Exudative lesion—beginning apical cavity	10 mm.	very marked	-10	
32.	Atelectasis. Mixed lesion	40 mm.	marked	-25	
33.	Obliterating pleuritis exacerbation	16 mm.	marked	-16	
34.	Exudative lesion	45 mm.	very marked	-45	
35.	Productive	27 mm.	moderate	-15	
36.	Exudative	30 mm.	very marked	-25	
37.	Mixed lesion—heavily infiltrated	60 mm.	marked	-50	
38.	Productive	40 mm.	moderate	-20	

TABLE I—CONT'D

NO. TYPE OF LESION	CUT. SENSITIVITY	POSTOP. REACTION	DESENSITIZATION	REMARKS
39. Mixed—new cavity	35 mm.	moderate	-15	
40. Mixed type	35 mm.	marked	-20	
41. Mixed type	20 mm.	moderate	-20	
42. Mixed type	40 mm.	marked	No test. Patient died.	
43. Empyema	30 mm.	very marked	none	Wound infection
44. Mixed type	30 mm.	marked	-20	
45. Productive	20 mm.	mild	+10	
46. Productive	15 mm.	mild	none	

individual's sensitiveness to tuberculin. For example, it would be practically impossible to express any great amount of antigen by collapsing a large open cavity with little perifocal infiltration, for the completeness of excavation has removed the antigen-containing tissue. Similar reasoning may be applied to the case where cavities are surrounded by scattered fibrosis. However, where the lesion is exudative and excavation is still in progress much antigen-containing tissue remains, and the sudden fall of a chest wall on such tissue may be disastrous in an individual where sensitiveness to tuberculin is great.

It will be realized, after an examination of Table I, that the above principles are relatively well borne out by the record of the cases presented. It will also be seen that parenchymal infiltration appears to play almost as important a part as the stage of the disease. The tendency on the part of those patients with much parenchymal infiltration to react more violently postoperatively than those with more scattered productive disease, is undoubtedly due to the presence of areas of relatively new or caseous disease which have been masked by dense fibrosis and pleural thickening. Furthermore, it is often impossible to estimate age of disease from x-ray films. The prints of x-rays of Case 34 (Figs. 1 and 2) presented here substantiate such views. The case in question has all the appearances of the so-called "good chronic," and if the films alone were seen, such a diagnosis would be made with perfect justification. However, when the previous films are reviewed, it will be seen that the patient's present state had evolved in less than seven months, and that such a disease process must be classified as recent, rather than old. It thus appears from the analysis of these cases that any area of dense consolidation, if present in association with a high state of allergy, must be looked upon with more than usual concern. There are several instances reported here of soft and recent disease associated with varying states of sensitiveness. The reaction which resulted in these cases depended to a large extent upon the amount of tuberculin expressed. Thus, Case 39, with relatively new disease and a rather high degree of hypersensitiveness, had little reaction when small stages were used while

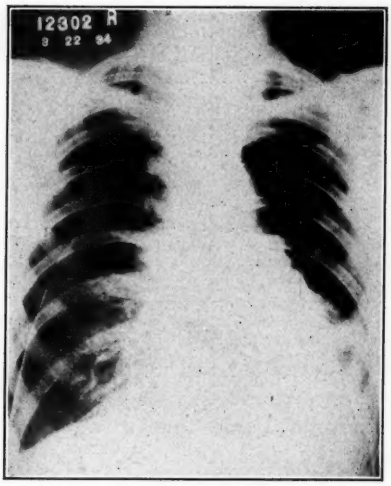

Fig. 1.

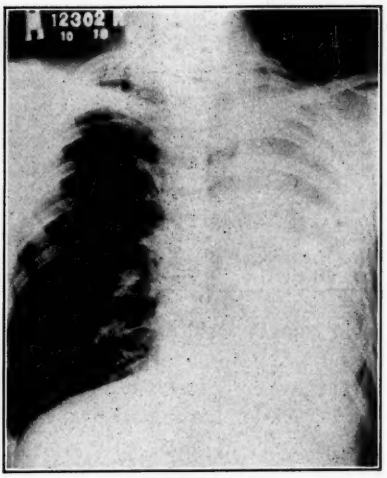

Fig. 2.

Fig. 1.—Film of patient who had had bilateral pneumothorax for two years. The left lung has reexpanded, the right is reexpanding. Sputum is negative.

Fig. 2.—The same patient as shown in Fig. 1, seven months later. There was a sudden temperature elevation, the sputum became positive. Note the marked atelectasis and appearance of chronicity.

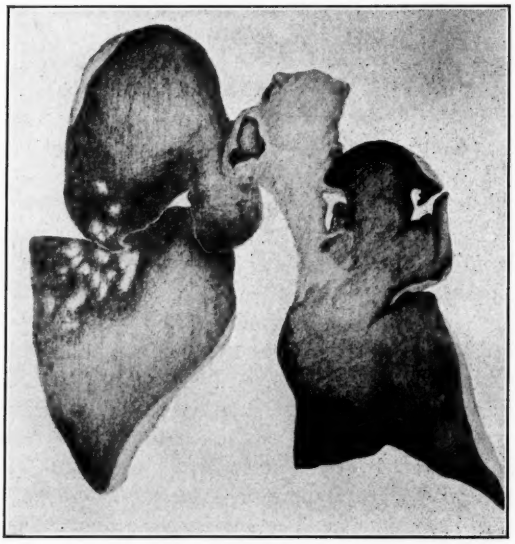

Fig. 3.—Drawing of a lung removed at autopsy from a young man dying of autotuberculinization. Note the area of consolidation about the old lesion in the contralateral lung.

Fig. 4.—Photomicrograph (×70) of one of the old lesions in lung shown in Fig. 3. Note limited edema and hemorrhage about fibrous tubercles.

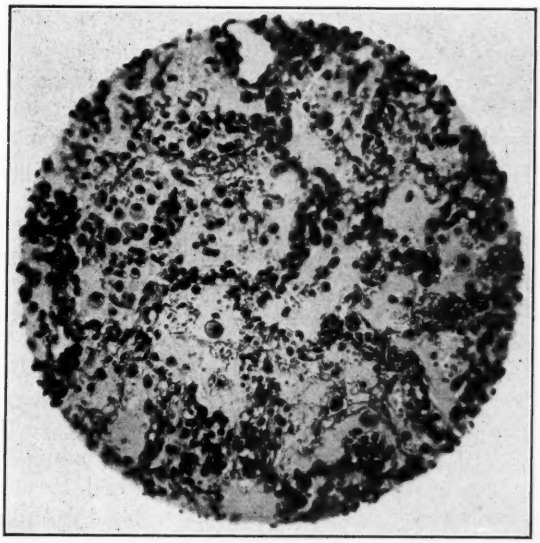

Fig. 5.—Photomicrograph (×500) of area of edema shown in Fig. 4. Note edema, hemorrhage, fibrin and large phagocytic cells, and swelling of alveolar walls. Note also similarity of the picture to that seen in tuberculin poisoning in animals.

Case 31, with a lesser degree of sensitiveness, reacted violently when a full first stage was done. It seems safe to believe that had Case 31 been more tuberculin-sensitive, the outcome would have been fatal.

It is thus apparent that the two factors, namely, the amount of tuberculin expressed, which seems to be dependent upon the type of disease, and the individual sensitiveness to tuberculin, go hand in hand in giving rise to reaction.

Coryllos[7] in 1934 doubted the soundness of the hypothesis of autotuberculinization. He based his opinion on the assumption that the blood and lymph flow from the areas of collapsed lung tissue was diminished or actually stopped. It is granted that some suppression may occur, but it is further contended that many factors must play a part in the amount and degree of hindrance to the normal lymphatic and blood circulation. That tuberculin must escape from such collapsed areas is borne out by Case 12 (Fig. 3), a drawing of the lung of which is shown, as well as photomicrographic pictures (Figs. 4 and 5) depicting the typical perifocal reactions seen constantly in experimental animals dead of tuberculin overdosage. Coryllos shows an x-ray film demonstrating this perifocal reaction in one of his patients in the same article in which he disputes such occurrences.

The point now arises as to the specificity of the tuberculin reaction and the ability of the antigens other than tuberculin to desensitize the tuberculous individual.

The researches of Long and Seibert[8] over a number of years have been productive of a highly purified tuberculin. They have shown the substance to be a protein, although the exact nature of its protein molecule is still unknown. On purely theoretical grounds, it might thus be said that tuberculin, being a protein, is also an antigen. Support for such an assumption has been given recently by Seibert.[9] She has demonstrated that tuberculin when injected over a period of time into the experimental animal, is capable of eliciting the Arthus phenomenon and gives rise to the formation of precipitins in such animals. Thus the antigenicity of tuberculin appears well established.

From time to time over the past twenty years, doubt has been cast as to the necessity of utilizing the specific antigen in desensitization experiments. Rich, Jennings and Downing[10] in 1933 found that it was possible to desensitize rabbits sensitive to pneumococcus Type I by the use of large doses of horse serum, as well as bacterial antigens, given intravenously in large doses. More recently, Rothchild, Freidenwald, and Bernstein,[11] working in Rich's laboratory, have stated that it was possible to desensitize tuberculous animals to relatively large test doses of old tuberculin by the use of various substances, notable among which was O. T. broth control. We have been unable to confirm their findings in work done recently in our laboratories, and which will subsequently be published. It was found, for example, that nonspecific antigens (horse

serum, egg white, bacterial suspensions, histamine) had no effect upon the tuberculin reaction when given in graded small doses, or in single large doses. It may be cited that there was apparently a rather high morbidity among the animals from which their conclusions were drawn, and the interpretation of results from such experimental media must be guarded. That states of anergy do exist in both tuberculous human beings and laboratory animals is not denied, but that such states can be induced by antigens other than tuberculin needs further experimental proof.

Chart V represents a small control series of patients who were tested during the course of these experiments. Patients A, B, and C represent cases of active pulmonary tuberculosis who suffered wound infection,

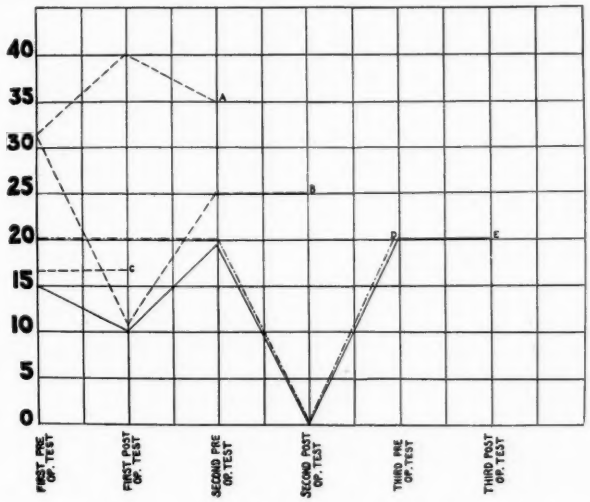

Chart V.—*A, B,* and *C* represent patients with wound infection following thoracoplasty.

Case A. Infection following first-stage thoracoplasty.

Case B. Infection following second-stage thoracoplasty.

Case C. Infection following first-stage thoracoplasty.

Case D. Demonstrates lack of reaction when first-stage thoracoplasty was over normal lung tissue, and marked reaction and loss of sensitivity to test dose of tuberculin when second stage was done over exudative lesion.

Case E. Productive disease under first stage. Exudative under second stage following a spread in this area after the first-stage thoracoplasty.

with temperatures running from 103° to 105° following thoracoplasty. It will be seen that Cases A and C show no fall in skin sensitiveness after the first stage, at which time there was no infection of the wound but a moderate postthoracoplasty reaction. There was in this case little diseased lung tissue under the second-stage area, and the stage was done merely to reinforce the first-stage operation. The temperature rose to 104° on the second postoperative day, and it was subsequently necessary to open the wound. It will be noted that following the second stage

there was no change in the skin test. Case D is that of a young woman with no disease under the first-stage area, but rather extensive exudative disease under the second-stage area. It will be seen that when there was no reaction following the first stage, at which time the temperature rose only to 100° by rectum, the skin test remained unchanged. This may be compared with the complete desensitization following the second stage, at which time the reaction was acute with corresponding temperature elevation, slight dyspnea, and cyanosis.

Case E corresponds rather closely to that of Case D with the exception that following the first stage there was a spread into the second-stage area. It will be noted that while following the first stage there was little change in the skin test, after the second stage there was complete disappearance of skin sensitivity to 0.1 c.c. of a 1:1000 dilution of O. T.

These cases, while few in number, appear to lend support to the premise that the reaction is a specific one. The temperature elevation in most instances in the three cases having infected wounds was as high or higher than the average postthoracoplasty reaction case; prostration and other evidences of being acutely ill, however, were not as marked as in the autotuberculinization cases. The two Cases D and E bring forth the principle that thoracoplasty per se causes little reaction and little change in skin sensitivity when done over normal lung tissue (Case D), or very old fibrous tuberculosis (Case E), but that the reaction may be acute and desensitization practically complete (to the test dose) when the underlying lesion is exudative, or mixed in type. Further proof of this last statement may be found in the absence of any type of reaction following extensive thoracoplasty done for chronic nontuberculous empyema which might be compared with the type seen following a limited thoracoplasty over an exudative tuberculous lesion. While postoperative shock may be marked in the former, the postthoracoplasty reaction is not seen.

While it is not desired to discuss too fully the question of specificity of antigen in desensitization, the statement, handed down from one textbook to another and by word of mouth, that the acute infectious diseases desensitize the tuberculous individual, needs clarifying. In rebuttal of such views, the work of Nalbant done on children in the children's unit at the William H. Maybury Sanatorium is offered. Nalbant[12] found that there was no loss of cutaneous hypersensitiveness in 175 children ill with childhood or adult type tuberculosis, following these acute infectious diseases: measles, mumps, chickenpox, pertussis, and scarlet fever. He includes in his paper a series of children tested by Chadwick[13] with similar results.

In the conclusion of this discussion we wish to make clear that we do not believe that autotuberculinization is the only cause of postthoracoplasty morbidity and mortality. There are other causes, notable among which are, mediastinal shift and flutter, paradoxic breathing, and

atelectasis resulting from retained secretion in the bronchus. In a recent review of a small group of cases who died following thoracoplasty at the Herman Kiefer Hospital, it was found that approximately 30 per cent gave evidence of autotuberculinization. The surgical literature dealing with thoracoplasty deaths has laid many deaths to cardiac failure. Hedblom[14] in an article published posthumously stated that 20 per cent of 230 collected cases died from cardiac failure. With the premise that cardiac failure causes death following thoracoplasty, except in the occasional instance, we cannot agree. The physician has been prone to lay at its door many instances where the true cause of death was unknown.

Recently Pinner and Brosius[15] have found evidence from autopsy material indicating that in some cases at least signs of chronic myocarditis were present. It is felt that it is fair to say that patients with this amount of myocardial damage might not tolerate well the postthoracoplasty reaction. On the other hand, without a control series of a similar number of patients dead of tuberculosis without surgical treatment and a group of general surgical patients (nontuberculous) who died following operative procedures, it seems that the attachment of the term cardiac death to this postthoracoplasty group would hardly be justified.

There have been few operative deaths during the formative years of various surgical procedures which have not been labeled cardiac failure by pathologists and surgeons, only later to be proved otherwise by clinical and physiologic investigation. The case of intestinal obstruction before the understanding of the chloride balance is cited in example. We have come to believe that many of the deaths previously ascribed to cardiac failure were in reality due to severe autotuberculinization.

SUMMARY AND CONCLUSIONS

The postoperative course of the tuberculous patient undergoing thoracoplasty we believe to be closely associated with that patient's state of disease and degree of sensitiveness to tuberculin. It has long been known to the thoracic surgeon that relatively recent lesions constitute a greater risk when submitted to thoracoplasty than older ones. We feel that the mechanism of the reactions in such cases has been explained.

It is not contended that the total postthoracoplasty morbidity or mortality is to be explained on the basis of autotuberculinization, but it is felt that many of those cases which have previously baffled the clinician can be accounted for on this basis.

The reaction is one involving the union of antigen and antibody, and if severe enough, leads to desensitization of the host to the ordinary test doses. As has been shown by Opie[16] and by Culbertson,[17] complete desensitization is brought about only by a complete binding of total available antibody either by exactly the correct amount of antigen or by an

excess. In cases reacting more mildly, it is probable that the antibody has not been completely bound, and thus the skin test is reduced but not completely erased.

The reaction as evidenced by experiments on laboratory animals (still to be published) and by clinical tests is specific, and antigens other than the specific one do not desensitize.

The tuberculin skin test taken as an index of sensitiveness to tuberculin when correlated with the stage of disease offers a valuable guide in the extensiveness of the contemplated thoracoplasty.

A clinical entity, the postthoracoplasty reaction, has been described, and its basis related.

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DIAGNOSIS, TREATMENT AND PROGNOSIS IN TUBERCULOUS TRACHEOBRONCHITIS*

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THE term "tuberculous tracheobronchitis" signifies a specific mucosal and submucosal infection of the trachea or large bronchi and includes all types of lesions whether ulcerative or nonulcerative, hyperplastic or fibrostenotic. Tuberculous tracheobronchitis is probably almost always secondary to active pulmonary tuberculosis,[1] and in our experience the presence of this complication in its severer manifestations has resulted in a mortality of approximately 50 per cent in spite of active treatment that may have been directed at the parenchymal lesions. Because of the relatively grave prognosis, it is important that clinicians recognize the complication.

It is the purpose of this paper to describe the clinical and bronchoscopic characteristics by which these lesions may be diagnosed; to consider their relation to prognosis in pulmonary tuberculosis, with and without collapse therapy; and to discuss what means of treatment are available. With the exception of the important contributions of Eloesser,[2] Haslinger,[3] and Jackson[4] dealing particularly with the diagnosis of stenotic lesions, reports in the literature are few, and most of them describe patients with obstructive respiratory symptoms of varying severity (Schonwald,[5] Werner,[6] Packard,[7] and others[8]). The clinical recognition of the earlier manifestations of tuberculous tracheobronchitis and the importance of its occurrence as a complication of parenchymal tuberculosis appear to have been neglected.

CLINICAL SYNDROME

The clinical diagnosis is made on the basis of a careful history, one or more physical examinations, sputum studies, and a review of serial roentgenograms of the thorax. At the University of Michigan Hospital, a bronchoscopic examination is used in all cases to confirm or refute a presumptive diagnosis.[9] A combination of several of the signs and symptoms can be made the basis for a positive prebronchoscopic diagnosis of tuberculous tracheobronchitis in a high percentage of cases, if the examiner keeps the possibility of this complication in mind.[10] It is to be emphasized that the majority of the symptoms are

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not pathognomonic of specific infection but are based upon a mere reduction, perhaps very slight, in the size of the lumen in some portion of the lower airway. Suggestive symptoms in patients with pulmonary tuberculosis should, however, call for a complete study because of the probability that a complicating tuberculous tracheobronchitis is present.

The most constant symptom is wheezing or rattling. It may be inspiratory, expiratory, or both. It is usually intensified by moderately forced respiration and persists after thorough evacuation of sputum. The wheezing is usually noticed by the patient and often by others. It can be heard by direct auscultation or by placing a stethoscope near the corner of the mouth or at the suprasternal notch. Changes in the position of the patient may intensify or diminish the loudness of the wheeze. Persistent wheezing is not, however, pathognomonic. A small group of patients presenting only this symptom have no bronchoscopic evidence of tuberculous tracheobronchitis.

Some of these patients may have upper lobe bronchial lesions which cannot be seen bronchoscopically. In the others, however, there has been evidence of displacement from fibrosis, and the patients have had large amounts of purulent sputum which they were never able entirely to evacuate. The loudest wheeze of this type which I have heard was in a patient who had chronic fibroid phthisis with retraction and a superimposed exudative tuberculosis.

Bronchoscopic examination often graphically demonstrates the results of retraction: the trachea may be shifted far from the midline; the upper lobe bronchial orifice on the side toward which the mediastinum has moved may not be visible; the lower portions of the stem bronchi may be sharply angulated because of fibrotic change in the upper lobes. We have felt that the mechanical basis for wheezing in these patients was extrinsic, rather than in the bronchial mucosa.

Asthmatoid breathing frequently occurs in attacks. Subjectively, the patient experiences difficulty in getting air into, or out of, the lungs, and he may have a sense of substernal constriction. These patients are frequently wrongly diagnosed as suffering from asthma or "asthmatic bronchitis." Respiratory difficulty in true asthma is mainly during expiration; in tracheobronchitis it is predominant during inspiration. Although tuberculosis and asthma are occasionally coexistent, this combination is much less frequently met than pulmonary tuberculosis complicated by tuberculous tracheobronchitis.

The presence of persistent audible and palpable parasternal rhonchi is suggestive of obstructive lesions (McConkey and Greenberg[11]). Such rhonchi are best elicited by forced expiration. They may be heard diffusely over the chest, but when localized, the site of the lesion in the tracheobronchial tree may be predicted with reasonable accuracy.

Occasionally the development of sudden unexpected elevations of temperature in a patient having an otherwise normal clinical course may be the first obvious sign of tracheobronchial lesions sufficiently obstructive to cause a retention of secretions. The sputum may show great variations in amount from day to day, and the expectoration of an unusually large quantity may coincide with the disappearance of fever.

Patients with obstructive lesions frequently suffer from paroxysms of coughing with exceptional difficulty in raising sputum. There are often dyspnea and intermittent cyanosis on slight exertion in spite of an apparently adequate vital capacity (Case 1). The gradual or sudden development of severe asthmatoid breathing and respiratory difficulty may mean a nearly complete block of one or both stem

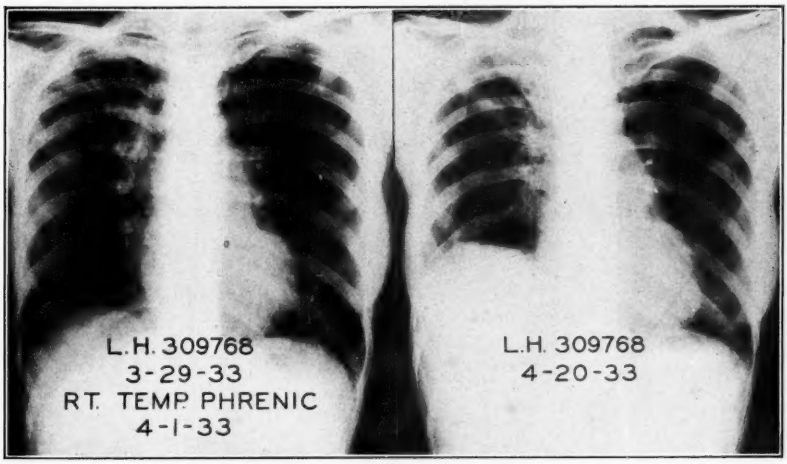


Fig. 1.—The occurrence of atelectasis of the right upper lobe following temporary paralysis of the right hemidiaphragm in a patient with tuberculous ulceration of the lower trachea, and ulceration and moderate stenosis of the right stem bronchus.

bronchi, or the trachea. Signs of massive obstructive atelectasis will develop if a lobar bronchus becomes completely blocked.

Several characteristics of the sputum in tuberculous tracheobronchitis are worthy of note. There may be excessively large amounts of sputum for the degree of pulmonary tuberculosis visualized roentgenographically (Case 4). The sputum may be exceptionally tenacious, mucoid or rubbery, difficult to evacuate, and often clinging to the bottom of the sputum cup when it is inverted. The purulent content is not necessarily high. Persistently positive sputum, in spite of an apparently quiescent parenchymal lesion, is a suggestive finding. Fibrin plugs or mucous bronchial casts containing tubercle bacilli are occasionally expectorated and are of great diagnostic importance in ulcerative lesions.

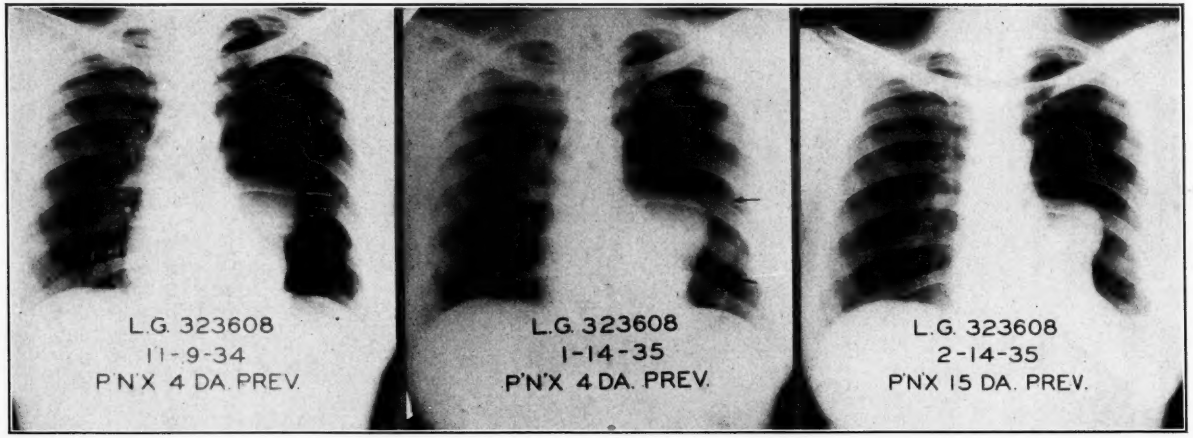


Fig. 2.—Three roentgenograms of a patient who had, bronchoscopically, hyperplastic and ulcerative tuberculous bronchitis of the left stem bronchus. The intermittent complete atelectasis of the *undiseased lower lobe* is well shown. The pneumothorax refills on each occasion varied from 300 to 350 c.c. of air, and the final pressures were slightly negative. Fluoroscopy on February 7, 1935, one week following pneumothorax refill, and one week prior to the roentgenogram of February 14, 1935, showed the lower lobe to be expanded. Since February 14, 1935, there has been consistent subtotal compression of the entire left lung.

Roentgenographic signs of tuberculous tracheobronchitis are not numerous, but they are important when present. Intermittent areas of atelectasis in serial roentgenograms of the chest are considered significant of bronchial obstruction. The phenomenon of sudden disappearance and later reappearance of pulmonary cavities which has been seen in some patients was probably also on the basis of intermittent bronchial obstruction. Barnwell, Littig and Culp[10] have called attention to the frequency with which partial atelectasis has followed phrenic paralysis, and with which total collapse of the lung has followed the induction of pneumothorax in cases of ulcerative tracheobronchitis (Figs. 1 and 2). Occasionally a tracheal or bron-

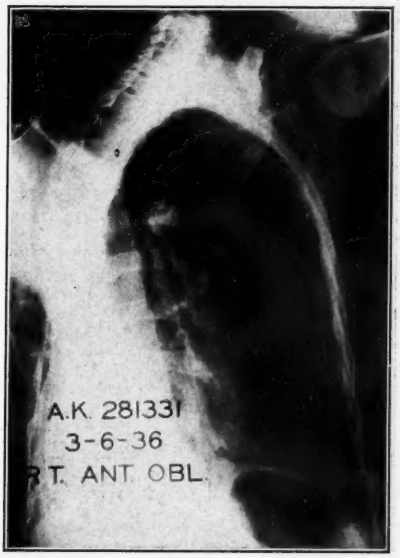


Fig. 3.—Stenosis of the stem bronchus visualized roentgenographically in the right anterior oblique projection using the Potter-Bucky diaphragm. The patient had had an extensive right-sided extrapleural thoracoplasty three years previously. The rapid development, recently, of respiratory distress, difficulty in raising sputum, and dyspnea, led to roentgenographic and bronchoscopic examinations. The stenosis as seen bronchoscopically showed no active mucosal disease.

chial narrowing is visualized, particularly in studies made with the Potter-Bucky diaphragm (Fig. 3). When the bronchoscopic examination is negative in spite of strong presumptive clinical evidence of tuberculous tracheobronchitis, brominol instillation may be employed to visualize stenoses "around the corner." Case 1 is illustrative.

CASE 1.—O. E., a white male, thirty-five years of age, chief complaints were productive cough, blood streaking, weakness, and fatigue. Tubercle bacilli were found in the sputum. Two years after the onset of symptoms, the patient was admitted to the University Hospital, where roentgenograms showed far advanced bilateral cavernous pulmonary tuberculosis, with the major involvement on the left side. For

about four months before admission, increasingly severe asthmatoid attacks and great difficulty in raising purulent viscid sputum had occurred. The patient wheezed continuously. There was dyspnea on the slightest exertion, and both dyspnea and cyanosis occurred during attacks of coughing. The vital capacity was 2,700 c.c. The patient was conscious of having to "push" during expiration. There were persistent parasternal and paravertebral rhonchi. A left partial pneumothorax was induced after which the asthmatoid attacks were less frequent. Bronchoscopy did not reveal a sufficient cause for the patient's marked symptoms; no active tracheobronchial mucosal disease was visualized. The left stem bronchus formed nearly a right angle with the trachea and curved slightly upward. The left upper lobe orifice, although incompletely visualized, appeared normal. The patient's symptoms were so outstanding that we felt the necessity for bronchograms (Fig. 4). Dr. Carleton B. Peirce, of the Department of Roentgenology, described the bronchograms as follows: "The left

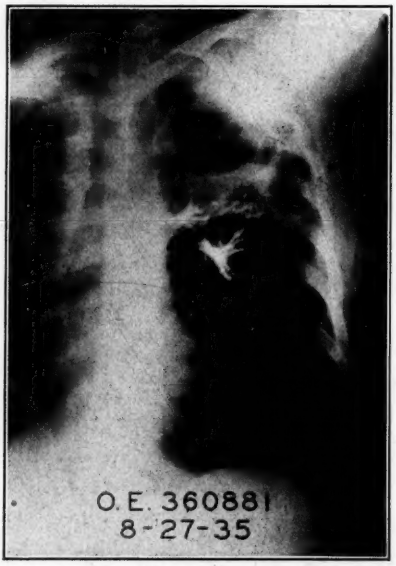


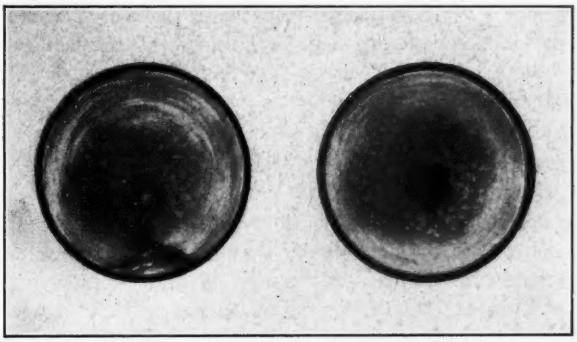
Fig. 4.—Bronchogram demonstrating a stenosis of the left upper lobe bronchus which could not be visualized bronchoscopically. The torsion of the left stem bronchus is well shown.

upper lobe bronchus presents incomplete filling with irregularity and constriction of the proximal portion, particularly the ascending branch leading toward the area of cavitation in the upper lobe. Torsion of the left stem bronchus is demonstrated. This bronchus leaves the trachea almost at a right angle, with the concavity upward." A similar condition was possibly present in the right upper lobe bronchus, but the patient was too ill for further diagnostic procedures. Abandonment of the inefficient left pneumothorax produced no change in symptoms. The patient was discharged from the hospital at his own request, with a hopeless prognosis, and died several weeks later at home.

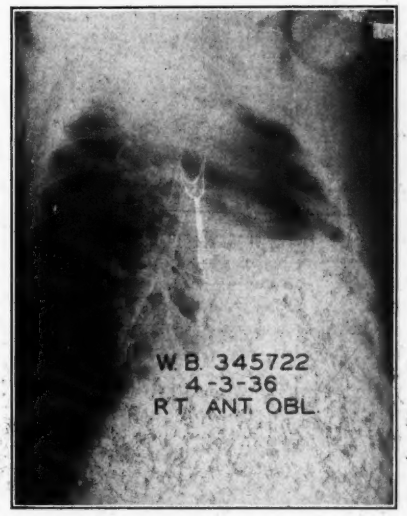
Radiopaque oil also has been used to visualize the extent and character of some bronchial stenoses which were too narrow to permit a bronchoscopic exploration. Such visualization occasionally has been a valuable procedure in determining both the advisability of dilatation and the direction in which bougies should be passed (Fig. 5).

BRONCHOSCOPY

At the University of Michigan Hospital, bronchoscopy has become a routine measure for the determination of a diagnosis of tuberculous tracheobronchitis.[9] The type and extent of the disease found serves as a criterion for treatment both of the tracheobronchitis and of the pulmonary parenchymal tuberculosis. Bronchoscopic examinations at intervals of several months may be employed to ascertain whether the



A. *B.*



C.

Fig. 5.—*A*, Bronchoscopic view of hyperplastic ulcerative and stenotic tuberculous mucosal disease of the left stem bronchus, showing the proximal end of the high grade obstruction which followed roentgen therapy. Tenacious purulent secretions are seen welling up through the narrowed orifice. Chemical shrinkage of the mucosa and mechanical dilatation of the lumen were necessary because of fever and paroxysms of cough. *B*, Increase in size of the lumen following chemical shrinkage and bougination. *C*, Bronchogram of the same patient in the right anterior oblique projection (retouched). The stenosis extends from 1 cm. below the carina to the left upper lobar orifice. Bronchiectatic dilatations are visible distally.

tracheobronchial lesions are progressing or regressing. Symptomatic treatment by the bronchoscopist is valuable in selected cases. The contraindications are advanced laryngeal ulceration; recent severe hemoptyses; hopelessly advanced exudative or pneumonic pulmonary tuberculosis, unless there are associated severe respiratory obstructive symptoms which need relief. A thorough bronchoscopic examination should be made, but the importance of gentle manipulation cannot be emphasized too strongly. When active obstructing mucosal disease is found, there should be no attempt to force the bronchoscope beyond the obstruction. In a patient recently bronchoscoped, a tracheal obstruction was discovered. The mere passage of a small bougie to measure the size of the lumen increased the mucosal edema and almost produced a fatal suffocation.

Bronchoscopically four types of lesions are distinguishable, two or more of which may coexist in the same patient:

1. The nonulcerative, nonstenotic type. The usual portal of entry into the mucosa is presumably by way of minute ulcers or mucous-gland-crypt tubercles, apparently caused by direct contact with sputum containing tubercle bacilli, according to the work of Bugher, Littig and Culp.[1] In the "nonulcerative" type of the disease, the minute ulcers are not visualized. The lesions seen bronchoscopically are submucosal infiltrations with occasional discrete tubercles. The overlying mucosa is not grossly ulcerated, but is congested, granular, edematous, and bleeds easily.

2. The hyperplastic type. The pathologic changes are entirely similar to Type I above, but are much greater in degree. There is submucosal proliferation and a piling up of tissue. Conglomerate tubercles or tuberculomas may be seen. Bronchoscopically these lesions cause an alteration in the contour of the trachea or stem bronchi, often decreasing the diameter of their lumina more or less greatly.

3. The ulcerative type. There is grossly visible diffuse or circumscribed loss of mucosa. Occasionally the ulcers extend to the cartilaginous rings causing a craterlike defect in the continuity of the airway. The ulcers are rarely solitary. Pure tuberculous, or mixed tuberculous and pyogenic, granulation tissue may be present. Frequently lesions which are predominantly hyperplastic show redundant superficial granulation tissue. Ulcers may not be visible bronchoscopically in these cases, but the presence of granulation tissue always indicates the loss of mucosal continuity.

4. The fibrostenotic type. All cases in which the lower airway obstruction is due primarily to intrinsic (endobronchial) scar tissue formation are included in this classification. The stenosis may be sharply localized; often an irregular scar tissue tunnel, one or more centimeters in length, is found. In many patients stenoses are accompanied by residual mucosal ulceration, with or without granulation

tissue. The pathogenesis of fibrostenoses is not always clear. We feel that they are usually due to the fibrotic healing of ulcerative lesions, particularly when circumferential. Thus they fall into the second group of v. Schrötter's[12] classification of stenoses, that of the mural (inflammatory stricture) stenoses. Eloesser[13] believes that the healing of extensive submucosal infiltrates may occasionally result in enough fibrosis to cause stricture.

The healing of ulcerative lesions by extensive scar tissue formation is not necessarily beneficial. Case 2 well illustrates the formation of a damaging fibrostenosis resulting from diffuse ulcerative tuberculous tracheobronchitis. The importance of repeated bronchoscopic examinations is stressed.

CASE 2.—D. W., a white woman of seventeen years of age, when first seen had an unstable bilateral apical pulmonary tuberculosis. The lesions progressed bilaterally when the patient was treated by bed rest alone, and a bilateral pneumothorax was eventually necessary for their control. The sputum became continuously negative for fourteen months. During a period of three weeks increasing difficulty in raising sputum developed, and there was a sudden onset of severe dyspnea and cyanosis. A fibrin plug was removed by direct laryngoscopy with complete relief. During the next four months the patient developed continuous wheezing, intermittent dyspnea, and difficulty in expectorating sputum which was tenacious and persistently positive.

The first bronchoscopy showed edematous tracheal mucosa. There were several oval areas of ulceration on the anterior tracheal wall, about 4 cm. above the carina. A piece of mucosa was removed and showed active caseating tuberculosis. The lower trachea was slightly narrowed by scar tissue. Symptomatic relief from wheezing and difficult expectoration lasted for several weeks. One month later a second bronchoscopy showed patchy hyperemia of the tracheal mucosa, but no ulceration. A new, sloughing, obstructive ulcer was seen in the right stem bronchus, 3 cm. below the carina. A portion of the slough was removed and showed tuberculosis microscopically. There was again symptomatic relief. Two months following the second bronchoscopy, a third examination showed the mucosa of the trachea and right stem bronchus to be intact throughout. There were slight roughening, edema and hyperemia, but no ulcers were seen. The patient was almost symptom-free for five months, and then again experienced difficulty in expectorating tenacious sputum. Extensive patchy sclerosis of the tracheal mucosa with narrowing was seen bronchoscopically. Both stem bronchi and all lobar orifices were stenosed by fibrotic infiltration and scarring. There was no visible ulceration. The patient died six weeks later, having had progressively severe respiratory difficulty, constant dyspnea, cyanosis, and terminal suffocation.

It is usually impossible to predict the type, extent or location of the lesions without bronchoscopic examination, since many patients present more than one type of lesion, and, in circumscribed minimal disease, there are practically no localizing signs. A "pure" lesion, that is, one consisting of a single type, occasionally can be diagnosed clinically with reasonable accuracy before the bronchoscopic examination is made.

CASE 3.—M. van B. was a white woman, twenty-five years of age. Moderately advanced left-sided pulmonary tuberculosis was first diagnosed two and one-half years

ago. The parenchymal disease was brought under control by the successive use of pneumothorax, temporary left phrenic paralysis and closed intrapleural pneumonolysis.

When tuberculosis was first diagnosed at the Michigan State Sanatorium, the patient had a persistent wheeze. She noticed that her sputum was more tenacious than that of other patients. It was occasionally positive, even after the parenchymal lesion was apparently quiescent. Over a period of one and a half years, the wheezing almost entirely disappeared, the sputum became less tenacious and negative for acid-fast bacilli. For three months preceding admission to the University Hospital, the patient again noticed wheezing which became constant and louder than at any previous time. Her vital capacity averaged 1,450 c.c. and in spite of this she had definite dyspnea when climbing one flight of stairs. Examination revealed an oral wheeze which persisted in both inspiration and expiration. There were sharply localized audible and palpable rhonchi just to the left of the sternum in the third and fourth intercostal spaces. She had some difficulty in raising small amounts of ordinary mucoid sputum.

The history was suggestive of active tuberculous tracheobronchitis. The abatement of symptoms, with the recent appearance of a slightly different syndrome suggestive of partial obstruction (slight dyspnea, difficulty in raising sputum and localized rhonchi), led to the clinical diagnosis of a stenosis of the left stem bronchus without active mucosal inflammation.

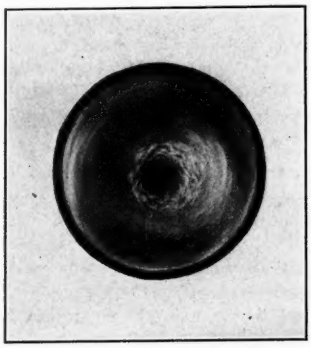


Fig. 6.—Drawing made at bronchoscopy of a smooth fibrostenosis showing no evidence of active mucosal disease in the left stem bronchus. Successful dilatation on two occasions was carried out, and the patient has been free of wheezing and dyspnea for eighteen months.

On bronchoscopy a stenosis of the left stem bronchus was seen about 2.5 cm. below the carina. It was a concentric narrowing, and the orifice was about 2 mm. in diameter. The mucosa was smooth and showed no evidence of active infection (Fig. 6). On two occasions, careful dilatation with soft bougies was carried out, the largest being No. 16 F. A small amount of pus was seen beyond the stenosis, and following the dilatations the sputum temporarily contained a few acid-fast bacilli. It has, however, now been consistently negative for eighteen months. The wheeze and dyspnea have disappeared. The vital capacity increased to an average of 1,750 c.c.

The following case illustrates some of the symptoms associated with tracheobronchial disease which is predominantly ulcerative.

CASE 4.—G. B., a white female, thirty-seven years of age, had an influenzal type of onset of tuberculosis six months before admission. Productive cough and ease of fatigue persisted. One month before admission, she noted a gradual onset of

wheezing and rattling in her left chest. Examination showed a persistent oral wheeze, present *after* evacuating sputum. A few fine posttussive râles were heard anteriorly over the left chest, and occasional rhonchi were heard both anteriorly and posteriorly on the left. The patient raised sputum easily, the amount varying from 40 to 60 grams daily, and always containing numerous acid-fast bacilli. The sputum was exceptionally tenacious and "rubbery" in character, and its purulent content was small. The roentgenograms showed a minimal left apical pulmonary tuberculosis. On the basis of a disproportionately large amount of tenacious, highly positive sputum, and in the absence of conclusive signs of obstruction (dyspnea, cyanosis, and difficulty in raising sputum), a clinical diagnosis of probable ulcerative tracheobronchitis without stenosis was made.

The bronchoscopic examination confirmed, in part, the clinical impression. Beginning about 3 cm. above the carina there were shallow ulceration, granulation tissue, and at least two tuberculomas. The mucosal change extended well into the left stem bronchus, the orifice of which was completely surrounded by ulceration and was sufficiently narrowed so that the passage of a 7-40 bronchoscope was not deemed feasible. In the trachea the ulcers were sharply limited to the left lateral and posterolateral segments. The surrounding intact mucosa was edematous and moderately congested. The bronchoscopist's opinion was that the disease was acute and exceedingly active. The mucosa was thoroughly shrunken with cocaine-epinephrine solution, and following bronchoscopy the wheezing and rattling were definitely diminished.

After a period of observation, roentgen ray therapy was instituted. Three days after the fifth treatment, the patient suddenly developed signs of a left lower-lobe atelectasis. It was felt that the roentgen therapy had stimulated congestion and edema sufficient to obstruct the left stem bronchus. In retrospect, this lesion was probably too active for roentgen ray therapy.

The bronchoscopic differential diagnosis is important. In arriving at a conclusion, the history, physical examination, and clinical impression are carefully weighed with the bronchoscopic findings. At the University Hospital we are at present opposed to taking a biopsy specimen if the preponderance of all available evidence supports the probability that the visualized lesion is tuberculous. Formerly, some patients in whom a biopsy specimen was taken developed an increase in their parenchymal disease, whether from aspiration of particulate matter or from the moderate bleeding that followed the removal of tissue could not be told. There is the further danger that a new tuberculous focus might be established should relatively normal mucosa be opened by the removal of a specimen. Myerson,[14] and Hammond and Warren[15] have reported no complications following biopsy. The latter routinely employ silver nitrate or the actual cautery to the bed from which the tissue has been removed. It is our custom to employ biopsy only when there is strong evidence that the disease seen bronchoscopically is not tuberculous.

The tuberculous mucosal lesions are found typically in the upper stem bronchi and lower fourth of the trachea, posteriorly and posterolaterally. Anterior tracheal tuberculous ulcers occasionally have been described. Lesions in the upper trachea may be secondary to active laryngeal tuberculosis. In the stem bronchi, the tuberculous

infection often involves the orifices of the upper and middle lobe branches, and if an exceptionally good view is obtained, involvement of the mucosa of these secondary bronchi occasionally can be demonstrated. Circumferential lesions are frequently seen in both trachea and bronchi, and the healing of such lesions may result in fibrostenosis. Unilateral bronchial involvement is almost always present on the same side as the more advanced parenchymal tuberculosis, and is more common than bilateral tuberculous bronchitis. In progressive lesions, repeated bronchoscopies will often show gradual encroachment upon the carina and the extension of the disease past the spur to the mucosa of the other stem bronchus. Lesions confined to the bronchi are more frequent than those occupying both bronchi and trachea. Tracheal lesions without accompanying bronchial lesions are not common. It is noteworthy that when one stem bronchus and the trachea are affected, the tracheal disease is frequently in the posterolateral quadrant, corresponding to the diseased bronchus. Tuberculous lesions rarely appear to be strictly localized or discrete, but tend to involve one or more centimeters of the lower air passages. The change from normal to abnormal mucosa is usually abrupt.

The more important conditions from which tuberculous tracheobronchitis has to be distinguished bronchoscopically are nonspecific inflammations and ulcers, erosion of lymphatic glands, neoplasm, syphilis, rhinoscleroma, asthma and, finally, the group of miscellaneous extrabronchial conditions, which may cause various degrees of tracheal or bronchial obstruction, the compression stenoses.

Diffuse congestive and inflammatory change affecting the entire tracheobronchial mucosa is in favor of nonspecific infection. Localized nonspecific mucosal congestion without infiltration is often found on the medial wall of the stem bronchus opposite the upper lobe orifice, probably the result of cough and expectoration. It may be difficult to differentiate nonspecific from tuberculous ulcers. In general, nonspecific ulcers tend to be discrete and are usually more shallow than tuberculous ulcers. They do not possess shaggy irregular edges, and there is rarely surrounding submucosal infiltration. They may be found in any portion of the tracheobronchial tree. Nonspecific inflammation and ulcers usually regress as the parenchymal tuberculosis becomes inactive and the cough subsides.

Peribronchial glands (often tuberculous) occasionally rupture into the lower air passages. Children are more frequently affected than adults. The onset of symptoms is usually sudden and is similar to that of foreign body obstruction. Violent paroxysms of coughing, livid cyanosis, and a feeling of suffocation are characteristic. A perforating ulcer, usually near the carina on the mesial wall of one of the stem bronchi, may be found bronchoscopically. The expectoration of disintegrated lymph node elements often mixed with blood, the

presence of a perforation in the characteristic location, and radiographic evidence of enlarged peribronchial glands aid in establishing the diagnosis.

Tertiary syphilis may be encountered in the lower air passages. The gumma in various stages of development may be found in any part of the tracheobronchial tree and may cause varying degrees of obstruction. The ulcers show little tendency to bleed when traumatized. Biopsy is usually necessary to establish the true identity of this lesion. Luetic lesions may heal with the formation of obstructive cicatrices, although these are no longer common.[16] They have no characteristic form. Extensive fibrotic disease at the tracheal bifurcation, however, is supposed to be more commonly syphilitic than tuberculous (Eloesser[2]). Evidence of tertiary syphilis in other parts of the body and a positive Kahn or Wassermann test are important confirmatory diagnostic aids. In cases of severe syphilis, secondary tracheobronchial lesions have been described by Sergent.[16] Clinically there is a febrile catarrhal bronchitis. Bronchoscopically there may be tracheal mucous plaques or a generalized rose-colored, exudative, confluent enanthem. The tracheobronchial lesions generally precede the appearance of the secondary skin rash and disappear without a residuum when specific therapy is employed.

Bronchiogenic carcinomas are usually encountered after the age of forty. Their presence may be suspected because of persistent cough, hemoptysis, dyspnea on exertion, pain in the chest, and loss of weight. In advanced carcinoma, secondary pulmonary suppuration is almost inevitable because of bronchial occlusion. Bronchoscopically, either a fungating obstructing mass which bleeds easily when traumatized or an ulcer with rolled indurated edges may be seen. There is often submucosal extension. There is no characteristic location. Carinal fixation from regional lymph node metastasis may be present. Lymphoblastomatous mucosal infiltrates may be occasionally encountered, and with these there is usually no ulceration. In all cases of suspected malignancy, a biopsy must necessarily be employed to establish a diagnosis.

Rhinoscleroma usually appears in the form of nodules in the mucous membrane of the nose and nasopharynx after the fourth decade of life. The nodules coalesce forming irregular, infiltrative masses in and beneath the mucosa. Ulceration is very rare. The process may be slowly progressive and eventually involve the tracheobronchial mucosa. The lesion is hard and flat, and the surface is dark brownish red. In the trachea the lesions are usually seen on the posterior wall. Fibrosis and occasionally annular excentric constrictions occur with healing; fibrosis may be accelerated by irradiation, but active lesions tend to recur. The final diagnosis is usually determined by special pathologic studies of a biopsy specimen.

There are no persistent gross changes in the tracheobronchial mucosa in asthma. Occasionally, however, if a patient is bronchoscoped during status asthmaticus, diffuse congestion and edema of the mucosa will be seen, giving the appearance of pink velvet. The orifices of all secondary bronchi are noticeably reduced in size and may close entirely on coughing, or on moderately forced expiration. Extremely tenacious sputum is usually present. The topical application of equal parts of 10 per cent cocaine and 1:1000 epinephrine hydrochloride solutions causes shrinkage of the mucosa and an increase in the bronchial lumen, which gives great symptomatic relief. The extensiveness of the mucosal changes and the peculiar swollen velvety appearance of the mucosa seen in asthma cases is against a diagnosis of tuberculous tracheobronchitis.

Any of the causes of compression stenosis may coexist with pulmonary tuberculosis and give rise to obstructive respiratory symptoms. The bronchoscopist should bear these possibilities in mind, forewarned by a careful history and roentgenologic studies. The compression stenoses have been completely described by Eloesser,[2] Haslinger,[3] and Jackson.[4] Anteriorly, the trachea may be compressed by an enlarged thymus, substernal goiter, dermoid, bony tumor of the sternum, abscess, or lymphoblastomatous infiltration. Posteriorly, compression may occur from tumors of the vertebrae or esophageal neoplasms. Aneurysms may cause compression in any portion of the tracheobronchial tree, and it should be remembered that lack of pulsation does not necessarily rule out aneurysm. In any compression, the tracheobronchial mucosa may be grossly negative, or it may be granular or ulcerated, depending upon the duration of the compression and the degree of infiltration by the compressing agent. Occasionally enlarged tracheobronchial lymph nodes cause a pressure narrowing of the trachea or bronchus without scar tissue stricture. This is apparently rarer in adults than in children and has been seen in only two cases in our experience. The bronchoscopic picture is often that of a semilunar indentation, with overlying smooth mucosa, on the posterior tracheal or mesial bronchial wall near the carina. Often there is little fixation, and the bronchoscope may be maneuvered past the obstruction by carefully depressing the bronchial wall.

PROGNOSIS AND THE USE OF COLLAPSE THERAPY

A diagnosis of tuberculous tracheobronchitis has been made in over 50 patients in the past six years at the University of Michigan Hospital. In this group, six were not bronchoscoped, but two of the six subsequently died, and the diagnosis was confirmed at necropsy. Twenty-four patients have been followed for more than one year, or until death.

We are now classifying patients with tuberculous tracheobronchitis into two groups depending upon the type of lesion as seen bronchoscopically. The classification has an important prognostic significance.

Group 1. Patients in whom there is gross ulcerative disease, fibrostenosis or both. Ulceration probably signifies grave disease; patients with ulcerative tracheobronchial lesions usually present more severe symptoms than patients with nonulcerative disease. The fibrostenotic healing of ulcerative lesions is not necessarily beneficial (Cases 2 and 3), since respiratory symptoms may persist and exacerbations of the parenchymal lesions may occur because of insufficient evacuation of sputum. Obstructive emphysema, or atelectasis and bronchiectasis often follow stenosis. In Case 2 the immediate cause of death was a tracheal and bronchial stenosis, proved by repeated bronchoscopic examinations to be the result of healing of diffuse tuberculous ulceration.

Of the 24 patients observed for more than one year, 17 have been classified in Group 1. Nine of the 17 (52.9 per cent) are now dead and none of the others has yet returned to complete health. Since nearly all of the patients with tuberculous tracheobronchitis have been referred from the Michigan State Sanatorium, an adequate comparison can be made with the mortality rate of the general tuberculosis population at that institution (see Table I).

A further analysis of the nine deaths shows that five patients died with symptoms of typical respiratory obstruction due to tracheobronchial disease. In the remaining four, the complication of ulcerative tuberculous tracheobronchitis appeared to be but one manifestation of the patient's poor resistance to tuberculosis.

Formerly it was customary to treat the parenchymal tuberculosis as indicated, regardless of the known presence of ulcerative or stenotic tracheobronchitis. Sixteen of the 17 patients in Group 1 eventually received some form of active collapse therapy for their parenchymal pulmonary tuberculosis (phrenic nerve paralysis, pneumothorax, multiple intercostal neurectomy, extrapleural pneumonolysis with paraffin filling, thoracoplasty or a combination of these measures). In approximately one-half of these patients, symptoms of tracheobronchial disease were not apparent until after collapse therapy was begun, and occasionally not even until after the parenchymal disease was quiescent. In the others, however, the symptoms or the diagnosis of ulcerative and stenotic disease antedated the use of any collapse measures. We have no evidence that collapse encourages the development of tuberculous tracheobronchitis.

In general, the results with regard to the tracheobronchial lesions have been equally poor whether the patient was treated by bed rest only or by collapse therapy, and whether the designated treatment was instituted before or after the appearance of symptoms of tracheobronchial disease.

TABLE I

COMPARISON OF DEATH RATES IN PATIENTS WITH AND WITHOUT ULCERATIVE AND STENOTIC TUBERCULOUS TRACHEOBRONCHITIS

PULMONARY LESION AT ADMISSION (NATIONAL TUBERCULOSIS ASSOCIATION CLASSIFICATION)	MINIMAL	DEATHS	MODERATELY ADVANCED	DEATHS	FAR ADVANCED	DEATHS	TOTALS	DEATHS
Michigan State Sanatorium patients who had no tuberculous tracheobronchial lesions*	104	0	363	6 (1.6%)	645	134 (20.8%)	1,112	140 (12.6%)
Patients with ulcerative and stenotic tracheobronchial lesions (Group 1)	0	0	8†	3 (37.5%)	9	6 (66.7%)	17	9 (52.9%)
Patients with nonulcerative tracheobronchial lesions (Group 2)	0	0	3	0	4	0	7	0

*Leslie and Anderson's[17] statistics for resident and discharged patients over a recent four and one-half year period. All patients with known tuberculous tracheobronchitis have been excluded from these figures.

†One of these patients had no roentgenologic demonstrable parenchymal disease when the diagnosis of tuberculous tracheobronchitis was first made; one had had minimal pulmonary tuberculosis when first seen. At the time they entered a sanatorium, however, both had moderately advanced pulmonary tuberculosis.

Because of the poor prognosis, we are not now recommending any type of collapse therapy for patients with the ulcerative type of the disease, unless subsequent bronchoscopic examinations show that there has been some tendency for the ulcers to heal without the formation of a damaging stenosis. The control of the parenchymal tuberculosis is not sufficient, since ulcerative disease frequently appears to be self-propagating when once established. Major surgical collapse seems to have no beneficial effect on ulcerative and stenotic tuberculous tracheobronchitis. A number of patients undergoing major thoracic operations have had undue respiratory difficulty with inability to evacuate sputum and a stormy postoperative convalescence.

In 1930 Eloesser[2] stressed the desirability of bronchoscopy in patients with suspected stenoses before proceeding with thoracoplasty. We are now inclined to believe that it might be wise for all tuberculous patients who are candidates for major surgical collapse to have the benefit of a preoperative bronchoscopic examination.

Group 2. Patients with nonulcerative and nonstenotic tuberculous tracheobronchitis do not present striking symptoms, and the clinical and bronchoscopic recognition of these lesions has been recent. After extended periods of observation we came to believe that nonulcerative tracheobronchial disease, as that in other parts of the body, was less active than ulcerative tracheobronchitis. Consequently, these patients are now being given the benefit of whatever type of collapse therapy may be indicated for their parenchymal tuberculosis.

Of the 24 patients who have been followed for longer than one year, seven belong to this group. None are dead and four have closed cavities and negative sputum following thoracoplasty. The remaining three have intermittently positive sputum; one had a pneumothorax and two had temporary interruption of the phrenic nerve. One of the latter showed an increase in the extent of her nonulcerative tracheobronchial lesions and later, definite improvement following roentgen ray therapy.

It is as yet impossible to predict what the course of patients with nonulcerative tuberculous tracheobronchitis will be, following control of the parenchymal tuberculosis. We feel that some will progress to ulceration and, perhaps, to stenosis. The relationship of the nonulcerative to the ulcerative and stenotic types of lesions is not entirely understood, nor are the reasons for the apparent dormancy of the disease in some patients with the latter development of activity and severe symptoms. At the present time careful clinical and bronchoscopic reexaminations can be our only guides, and until further data are available we cannot evaluate the unknown factors of specific tissue resistance and susceptibility to tuberculosis as far as they affect the tracheobronchial mucosa. It is apparent, however, that in patients whose tracheobronchial disease has reached the ulcerative or

obstructive fibrostenotic stage, the prognosis for life and cure of the pulmonary parenchymal tuberculosis is grave, and we are at present exceedingly pessimistic of our chances of successfully dealing with these unfortunate patients.

TREATMENT

Many therapeutic measures have been employed for tuberculous tracheobronchitis without success. When this condition was first recognized, the frequency of asthmatoid symptoms led to the symptomatic use of atropine, epinephrine, ammonium chloride, and autogenous sputum vaccines at the University of Michigan Hospital. The only favorable effect was an occasional temporary alleviation of symptoms. Change of climate, ultraviolet light, and repeated subcutaneous injections of tuberculin have been employed without lasting success. While tuberculous tracheobronchitis is now being recognized in its earlier and less obvious stages, the methods of treatment are as yet by no means standardized. Experience and the individualization of patients with regard to the type of their tracheobronchial and parenchymal lesions may aid in finding some effective means of dealing with this complication.

At present bronchoscopic treatment appears to offer the best possibilities for symptomatic relief. Patients with nonulcerative disease, particularly if it is hyperplastic, occasionally have disturbing wheezing and difficulty in raising sputum because the diameter of the tracheobronchial lumen is decreased. The chemical shrinkage of the congested and edematous mucosa with equal parts of 10 per cent cocaine and 1:1000 epinephrine hydrochloride solutions applied directly by applicator and spray, especially around and into the orifices of the involved lobar bronchi, has given symptomatic relief which often has lasted many months. The patients lose their wheeze to a large extent and are able to raise sputum with relative ease. This treatment has been found of value preceding thoracoplasty. Patients with impending suffocation can be temporarily greatly improved by the bronchoscopic removal of obstructing tenacious secretions, crusts, or mucous plugs. All that can be expected, however, is immediate palliation; advanced tracheobronchial ulceration *and* stenosis usually indicate a hopeless prognosis. Severe postbronchoscopic laryngeal edema has necessitated tracheotomy in three patients, and following this, low bronchoscopy and catheter aspirations were facilitated. All three, however, eventually died.

We have no set rule for the treatment of cicatricial obstructive lesions. Localized stenoses of the stem bronchi can be successfully and repeatedly dilated bronchoscopically (Case 3). There is often complete remission from wheezing and rhonchi, and from fever due to retained sputum.

CASE 5.—J. P., a white woman, thirty-five years of age, had an insidious onset of her tuberculosis seven years prior to her admission to the University Hospital. There were fever, weakness, and loss of weight. The sputum was positive for acid-fast bacilli. A left-sided pneumothorax had been attempted on two occasions without success. A left permanent phrenic paralysis had been produced. Admission roentgenograms showed a predominantly left-sided far advanced cavernous pulmonary tuberculosis, with fibrosis and retraction. There were multiple parenchymal scars in the right upper pulmonary field. The patient had noticed a persistent wheeze for one year, and for four months there had been definite increasing difficulty in raising from 25 to 40 c.c. of purulent sputum. Consequently, a bronchoscopic examination was performed prior to the contemplated thoracoplasty. A cicatricial stellate narrowing was found in the left stem bronchus about one inch below the carina and

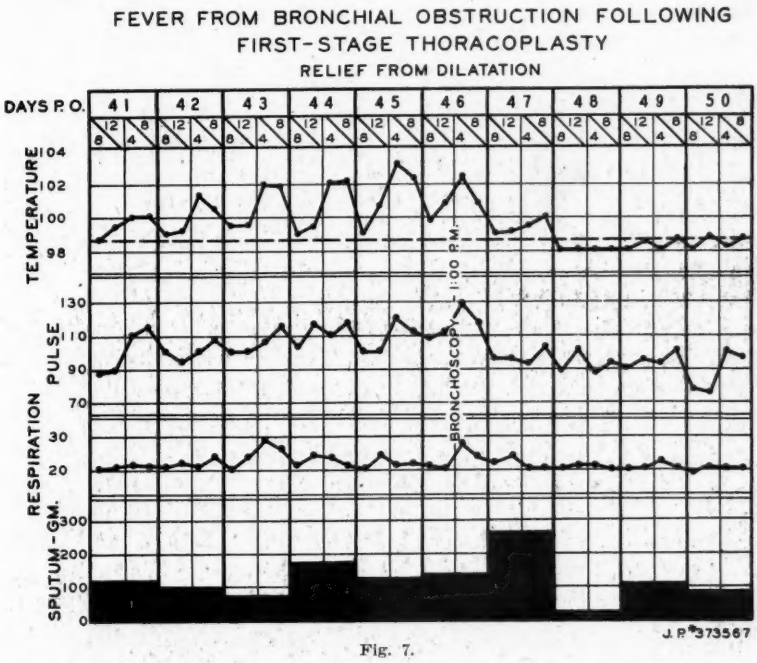


Fig. 7.

above the left upper lobar bronchus. A No. 14 bougie could be easily passed but not a 7-40 bronchoscope. The mucosa was congested but not ulcerated. The breathing space appeared adequate and thoracoplasty was believed to be not contraindicated. Following the first stage of the thoracoplasty, convalescence was very slow. There were intermittent fever, as high as 103° F., on the twelfth postoperative day, and difficulty in evacuating sputum. The sputum was heavily purulent and had increased from 50 c.c. to 200 c.c. in amount on the thirty-seventh postoperative day. From the forty-first to the forty-sixth day (Fig. 7), the fever became progressively elevated and the sputum was very tenacious and overlaid with froth. Bronchoscopy showed a high grade left bronchial obstruction, almost complete in expiration, and in part, evidently due to increased mucosal edema and congestion. Shrinkage of the mucosa and successive dilatations of the stenosis with Nos. 12, 14, 16, and 18 soft bougies were carried out. With each dilatation, large amounts of pus welled up

from beyond the stenosis. Within forty-eight hours the temperature became normal and remained so. The reaction to the second and third stages of the thoracoplasty was entirely without incident.

We much prefer not to dilate a stricture if the surrounding mucosa is obviously the site of active tuberculous infection, but at times dilatation is necessary (Fig. 5). It has been demonstrated that the passage of dilating instruments in the face of mucosal inflammation or ulceration can superimpose enough traumatic edema and congestion to cause rapid suffocation or a bronchiogenic pneumonia from retained secretions. Tracheal stenoses are much more serious than are bronchial stenoses, and in our experience attempted dilatation has led to uniformly bad results. If the surrounding mucosa is edematous, chemical shrinkage by spray is employed; the only indication for bouginage, or the forced passage of a bronchoscope, is impending suffocation which, however, may be increased by reactionary traumatic edema of the mucosa.

When a small, sharply-circumscribed ulcer is discovered in the tracheobronchial tree, local treatment with either the actual cautery or high frequency current may be an aid to healing similar to that reported in the larynx (Terry[18]). Such an ideal lesion occurs rarely, however, and the use of electrocautery in diffuse lesions is not without considerable danger.*

Roentgen radiation in divided doses has been given to twelve patients. This therapy was suggested by Jacox as a possible means of promoting healing by the early induction of fibrosis, similar to the reported action of irradiation in tuberculous lymphadenitis. Two references to the use of roentgen ray therapy in tuberculous tracheobronchitis were found in the literature. In Schonwald's[5] patient, the use of deep therapy had no influence on the fatal outcome. Vinson and Habein[8] fail to state whether or not the course of deep roentgen ray therapy which they gave was of value.

At the University of Michigan Hospital the roentgen ray beam is directed through two ports, one on the anterior, and one on the posterior surface of the body. The ports are either 10 × 10 cm., or 12 × 12 cm., and are centered over the lesions as described bronchoscopically. The anterior and posterior ports are treated alternately at weekly intervals. The filtration is ¼ mm. copper and 1 mm. aluminum; the voltage is 150 KV. (Villard circuit); the tube current, 25 MA.; the skin target distance, 50 cm.; the intensity of irradiation, from 34 to 39 r. per minute measured in air without back scatter. The dosage per treatment field varies between 200 r. and 300 r. Irradiation is continued until a definite skin erythema is produced, and this usually occurs in from six to eight treatments.

*Since writing this report, tuberculous mucosal lesions in several patients have been successfully treated by means of high frequency electrocauterization with special bronchoscopic electrodes. The method apparently has a well-defined place in the treatment of these lesions.

It must be emphasized that roentgen radiation is being used experimentally to a large extent at the present time, and we are not yet entirely convinced that the treatment is of value. No patient in the series has been observed sufficiently long to determine whether roentgen ray therapy is curative. Eight patients have had their series of treatments completed for three months or longer. In five of the eight patients bronchoscopic reexamination showed that the active lesions were undoubtedly improved, although two patients have shown further obstruction in a preexisting bronchial stenosis. In one of these (Fig. 5) frequent dilatations have been necessary because of distressing cough and fever from retained secretions. The tuberculous lesions in these patients have been the chronic hyperplastic type with submucosal tuberculomas, and the ulcerative type with granulation tissue. In the sixth patient, the lesions have become worse following irradiation, and there is now definite ulceration and right bronchial fibrostenosis, neither of which existed prior to roentgen ray therapy. The seventh patient had tracheal ulceration and partial obstruction from scar tissue. Bronchoscopy was not repeated following irradiation. She had almost complete symptomatic relief for two and one-half months following roentgen therapy, but during the past three months the severe cough, asthmatoid attacks, dyspnea and intermittent cyanosis have returned. The eighth patient (Case 4), had an acute ulcerative tracheitis and left bronchitis with moderate narrowing of the left stem bronchus. The untoward reaction to roentgen ray therapy which this patient suffered has been described.

From our limited experience we feel that patients with acute, extremely active ulcerative and hyperplastic tuberculous tracheobronchitis, and patients who already have a high grade fibrostenosis as well as active specific infection, do not offer good indications for roentgen ray therapy. Definite improvement has been seen in some patients whose tracheobronchial disease is chronic, and predominately hyperplastic and granuloulcerative. We have not yet had the opportunity of treating a patient presenting only excavating tuberculous ulcers.

SUMMARY

The term "tuberculous tracheobronchitis" signifies a specific mucosal or submucosal infection of the trachea and large bronchi, and is to be distinguished from "tracheobronchial tuberculosis" or tuberculosis of the tracheobronchial lymph nodes. Tuberculous tracheobronchitis is apparently nearly always a complication of active pulmonary tuberculosis. It has been shown that tuberculous tracheobronchitis can be considered a definite clinical entity, the diagnosis of which is based on rather characteristic symptoms, physical examination, and a study of serial roentgenograms. The importance of confirmatory bronchoscopic examination has been stressed, and the four types of lesions seen bron-

choscopically have been described. These are the nonulcerative, ulcerative, hyperplastic, and fibrostenotic; several types often coexist in the same patient. Of more than 50 patients with known tuberculous tracheobronchitis, 24 have been observed for at least one year or until death. The relatively grave prognosis in patients with ulcerative and stenotic lesions is shown by the 52.9 per cent mortality in this group. The apparent ineffectualness of pulmonary collapse therapy in this particular group has been demonstrated. Various methods of treatment of tuberculous tracheobronchitis have been discussed. In selected cases, bronchoscopy has given symptomatic relief. In five cases with chronic hyperplastic and granuloulcerative tracheobronchial mucosal tuberculosis, the use of roentgen ray therapy has apparently resulted in a regression of the lesions.

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DISCUSSION ON "THE TREATMENT OF PULMONARY TUBERCULOSIS BY MEANS OF AMBULATORY ARTIFICIAL PNEUMOTHORAX," J. A. MYERS; "A CONTROL GROUP FOR STUDYING THE END-RESULTS OF THORACOPLASTY: AN ANALYSIS OF THOSE PATIENTS REFUSING OPERATION," S. O. FREEDLANDER AND S. E. WOLPAW; "STUDIES ON TUBERCULIN HYPERSENSITIVENESS. I. THE RELATION OF HYPERSENSITIVENESS TO TUBERCULIN TO THE POSTTHORACOPLASTY MORTALITY," W. M. TUTTLE, E. J. O'BRIEN, AND EVARTS A. GRAHAM; "DIAGNOSIS, TREATMENT, AND PROGNOSIS IN TUBERCULOUS TRACHEOBRONCHITIS," PAUL C. SAMSON; "PARAFFIN PACK AND THORACOPLASTY IN THE TREATMENT OF LARGE APICAL CAVITIES," JEROME R. HEAD.

DR. EDWARD S. WELLES, Saranac Lake, N. Y.—I think this has been a very interesting group of papers, and it is a little difficult to know what conclusions to draw from some of them.

With reference to Dr. Myers' paper on ambulatory treatment with artificial pneumothorax, it is a little puzzling to me, coming from Saranac Lake, to hear of his great difficulty in getting patients to go to bed and take bed rest, to have to have so many patients as ambulatory ones, especially some of the ones he showed on the screen that obviously should be in bed. I suppose the difference is in our viewpoint. I live in a tuberculosis community; he lives in a city. By the time the patients get to us, they have been through the arguments with their doctors about whether they are going to go to bed or not, and if they come to Saranac Lake at all, they are willing to go to bed. But certainly, patients who are as sick as some of those whom he shows in his pictures, we would not have up as ambulatory patients. I fully appreciate his difficulties in dealing with patients that have not yet grasped the full significance of tuberculosis, and if his experience is typical of a large part of the country, it certainly seems that more education is necessary.

We feel that we are getting patients more and more educated to grasp the essence of treatment of tuberculosis, but apparently a great deal more is necessary to be done. It seems in one or two of his cases that pneumolysis might have been done if it had been attempted very carefully. However, one cannot say that without looking into them. He said that the cases had been looked into and pneumolysis found impossible to do.

We find more and more as we go on doing pneumolysis that there are fewer and fewer cases in which something cannot be done. Our percentage of complete collapses is pretty high.

I think Dr. Freedlander's figures are extremely interesting and valuable to us, but I wonder if Dr. Freedlander took home the charts that he has shown us, and put them up in his clinic so that the patients who are having thoracoplasties proposed to them might see them; it might cut down that large list of refusals. There again education in tuberculosis is lacking, because in our community and in other communities in New York State and in Vermont with which I am associated, we do not have this high percentage of refusals. Not that our results are so astoundingly good with thoracoplasty, because they are not. It is simply that we have been at it a long time, and the patients seem to be educated to take what we offer them; we have more difficulty turning down patients for thoracoplasty than we do urging them to have it. We have to turn down the ones that we do not think are capable of standing the operation. We have very, very few, throughout the year, that we advise to have thoracoplasty, whom we cannot persuade to accept it.

I think Dr. Tuttle's paper is going to be of very great significance to all of us. This question has come up and we have been thinking about it, but we have not worked it out. We have not done any experiments, as Dr. Tuttle has. I have

talked this over with Dr. Lawrason Brown many times. We find that the patients who die from thoracoplasty, die after their second-stage. I do not know what has been the experience of the rest of the surgeons in that, but we do not often lose a case after the first-stage of thoracoplasty, and the first-stage is undoubtedly the most difficult. Getting out the first, second, third ribs, way up in the top of the shoulder, is a much harder operation than any subsequent one, but still we do not lose patients from that. Our more or less routine operation is to take the first three ribs the first operation, then from there on we go in graded stages as we think best. The patients who die from thoracoplasty die after their second-stage, but we do not operate for the second-stage until the chart looks all right.

We feel that the pulse rise is probably due to shock, but the temperature rise does seem to be due to tuberculinization. We let that go down, let the pulse get level, and then do the second-stage; shockingly and unexpectedly some of them die, and Dr. Brown has assured me that we are then operating at the wrong time, from the standpoint of tuberculinization. That is his theory. He has not proved this. He does not hold to it absolutely, but he has had the idea that there must be a tuberculin reaction that we produce at the first operation, and that our two-week interval between operations, at which time the patient seems all right on the chart and seems all right by physical examination, may be wrong, because that may be just the bad critical time with the tuberculin.

I am sorry to say that I cannot get the answer to this question from Dr. Tuttle's paper, but I should like to talk to Dr. Tuttle. I talked to Dr. Graham this morning and he says he thinks he understands some of the features of it that I did not quite get from the paper. The idea is certainly there, but I still do not know the answer. Possibly more experimentation can be done.

I did not get from Dr. Tuttle's paper whether his results of this experiment had given him a conclusion as to how to manage the second-stage of his operation: if he had changed his time interval as the result of it, if he was going to, or if he had any definite thoughts about that. It certainly is very important. There is something wrong somewhere.

There is just one point I wish to bring out in addition to that. I think Dr. Head's results were splendid. I have never been an advocate of paraffin packs and do not use them, having always felt that I could accomplish as much with thoracoplasty as I could with paraffin. The results are very good. There is just one thing about his finished x-rays after operation. They all show no evidence of cavity, and he says that the sputum is negative for tubercle bacilli. I have come to disbelieve that conclusion. I do not mean that I disbelieve Dr. Head, but that x-rays that look like his do not show any cavity; if an x-ray is taken somewhat later with a Bucky diaphragm, or with a direct intentional overexposure, very often cavities do show up which do not show in these plain x-rays, and often sputum is negative for a period of time following an operation, which later begins to increase in amount and becomes positive again. The bane of my life in the last two or three years has been the return of apparently good results into the bad result class, the coming back of these patients that were sent out with pictures like this, not showing cavities, and with sputum reduced almost to nothing. They come back with long cigar-shaped cavities. Undoubtedly, you have all been seeing this. They come asking for reoperation. My very short paper, which was read by title, is on this subject; it is a desperate problem to know how to handle. I think some of these cases ought to be let alone. The burden of my paper is not to try too hard to close these noncollapsible cavities, for that is what I think some of them are. Just a word of warning: Do not become overoptimistic by the first picture. Some of these pictures may be very late, but all we can tell as we see them here is that this is the case before operation, and this is the one after opera-

tion, and frequently that one shown after operation is very soon after operation and shows no cavity. Let us not be too optimistic until we see the film taken at a considerably later period, with different types of technique that penetrate and bring out the cavity.

DR. HARRY R. DECKER, Pittsburgh, Pa.—I should like to speak with reference to Dr. Head's paper. I can see indication for plombage or paraffin pack in some cases where one might rather choose a thoracoplasty as a method to accomplish perfect results. I am thinking particularly of the United States war veterans, a rather tough bunch to handle in connection with the disease of tuberculosis. I have been able to sell several of them, who have point blank refused thoracoplasty, paraffin pack as a trial measure. I have had one unfortunate result from paraffin pack. An individual developed a staphylococcic septicemia, approximately seventy-two hours after the induction of the pack for a large apical cavity. There was no wound infection superficially and none in the musculature as far as we could judge, though we did not get a postmortem examination. There probably had been a contamination from the cavity underneath the pleura.

I did have a very fortunate result from a paraffin pack in a patient with basilar tuberculosis who came to me following a trauma, in which he had had a severe fall and injury of his costal lower ribs and lower thorax. He developed a rather exsanguinating hemorrhage, and bled to 42 per cent at this time. Transfusion and a phrenic paralysis controlled the hemorrhage, and he recovered from that attack. He had not known that he had tuberculosis, and it was at that time that he was apprised of it, and in the subsequent few months, he carried on with that knowledge at a reduced pace. A year later he had a similar fall. He had undertaken light work, struck the lower part of his chest again, and developed another severe hemorrhage. Because the bleeding was continuing, his condition did not justify doing thoracoplasty or waiting for it. I introduced, therefore, a 500-gram paraffin pack at the base of this lobe, and it controlled his bleeding perfectly. This man is still carrying this pack and has not bled since. As far as the tuberculosis goes, it does not seem to have arrested it completely. Though he is sputum-negative, there has been a little extension in the upper part of the lower lobe in the right lung. I think this man, ultimately, is a subject for thoracoplasty.

DR. EVARTS A. GRAHAM, St. Louis, Mo.—I wish to speak particularly about the work of Dr. Tuttle. Dr. Tuttle began this work a little more than three years ago, while he was with us in St. Louis. The plan at that time was to attempt to find out, if it would not be possible to determine a little more exactly than we were able to, the good risks and bad risks for thoracoplasty, on the basis of their sensitivity to tuberculin. Dr. Tuttle had come to us with a very considerable knowledge and experience in immunology, and he seems to be a very happy choice to undertake this problem. He had worked rather extensively with Long, in problems of immunology in tuberculosis. I mention all of this, because you may think that Dr. Tuttle is merely a surgeon. He is more than that. His work, therefore, carries some weight when he speaks about immunologic problems.

Of course many deaths after thoracoplasty are due, very plainly, to an extension of the tuberculosis. I have never held to the opinion that the prevailing idea is correct, that patients die after thoracoplasty operations in the first few days from heart failure. That idea is based merely upon the fact that these patients develop pulmonary edema. There is no other reason whatever for assuming that these patients die of heart failure, and yet all through the literature, particularly the European literature, the major cause of death after thoracoplasty operations is set down as heart failure. One finds in collected statistics in continental Europe that

approximately 80 per cent of the deaths from thoracoplasty in the first few days are ascribed to heart failure. As a matter of fact, I think that all of the evidence is against such an idea.

Dr. Tuttle, in carrying out one phase of this work, subjected patients before thoracoplasty operations to as careful heart examinations and tests of heart function, and so forth, as can be done. These patients, of course, are young individuals, comparatively speaking. They are not the class of patients who are likely to have myocardial heart disease, and as a matter of fact, the tests indicate that they are not victims of cardiac disease. Merely because they develop pulmonary edema is, therefore, no indication whatever of the fact that they die primarily of myocardial disease.

We hoped that by carrying out extensive tests of tuberculin sensitization on a series of patients, we might be able to divide the sheep from the goats. Dr. Tuttle has extended this work after leaving us and going to Detroit, and when you have an opportunity to read his paper I am quite sure that you will all feel that he has made a most important and significant contribution to our ability to prognosticate which patients are desirable for thoracoplasty and which are not. I do not mean to say this problem has been settled. I do not mean that by any simple minor skin test, one can tell with a hundred per cent degree of accuracy that a patient will go through a thoracoplasty operation or not, but I think that the information which he has gained will prove to be exceedingly helpful.

One might think that if this phenomenon is, at least partly, dependent upon the tuberculin sensitivity of the patient, he might look forward to a day when, perhaps by desensitizing such patients, he might eliminate this factor as one to be feared after thoracoplasty, and, thereby, greatly reduce still further the operative mortality. Unfortunately, however, this does not seem to be the case. Rich and McCordock, a number of years ago, showed rather conclusively, I think, that immunity against the disease tuberculosis is quite a different phenomenon from the reaction to tuberculin; that experimental animals, for instance, may go on and die from their tuberculosis, despite the fact that they have been completely desensitized to tuberculin. It does not seem to me, therefore, that there is much promise in the idea that we might at some time be able to get these patients into a condition more suitable for thoracoplasty simply by desensitizing to tuberculin, except so far as it relates to the fact that patients can die of a tuberculin reaction itself. Possibly some means may be found of saving that group, but it cannot be expected that desensitization to tuberculin will save the patients who die merely from an extension of the disease.

I wish to ask Dr. Samson a question about his work. I was not entirely clear from his presentation whether or not he regarded atelectasis as a favorable or an unfavorable development in tuberculosis. I got the impression that he regarded it as an unfavorable development in tuberculosis, and this was a rather startling idea to me, because I have had the opinion which I thought was rather general, that atelectasis was an exceedingly favorable development in pulmonary tuberculosis.

DR. HOWARD LILIENTHAL, New York, N. Y.—I have been very much instructed and rather thrilled by the exposition here of reasoning from statistical charts, which usually leave me a bit cold. I agreed perfectly with what Dr. Welles said, and, in fact, before he spoke I suggested to Dr. Bettman, beside me, that certain of these charts should be placed in the hands of patients whom we think ought to be operated upon and who refuse. You know how convincing figures may be, even though they may seem to prove contradictory things. In insurance companies figures do not tell lies, and there are certain companies which maintain that

they can pick out from passers-by in a very populous place, people who look like good risks, and insure them without a physical examination. Certainly, statistics do good in insurance matters, and I think they may be clinically useful as well.

Another word about the second-stage deaths concerning which Dr. Welles spoke. I believe that the danger of the second or lower rib stage is in doing too much at a time, since the amplitude of respiration is much greater, as you know, in the lower chest than it is in the upper, and the danger of tuberculinization, as I used to call it and probably shall continue to call it after what we have heard today, is much greater with a greater degree of collapse than it would be with a smaller one.*

DR. LEO ELOESSER, San Francisco, Calif.—Whereas Dr. Myers' paper may not be of paramount importance in this country as yet, it is of very great importance, I think, in those countries in which the number of patients greatly exceeds the number of available beds. It was interesting in Russia to see patients who had no bed rest. There is practically no tuberculous patient in Russia who gets twenty-four-hour bed rest in our sense. They are all more or less out of bed. I am not sure that they are not in a little better shape than our patients. I am not sure that we are not overdoing the twenty-four-hour bed rest.

Dr. Samson's paper was of great interest. I wonder whether some of these stenoses occur after thoracoplasty rather than before. The bronchoscope is not an absolutely diagnostic instrument. We must recognize that, except in circular strictures, it is not always possible to say, when we introduce a bronchoscope, whether with the instrument itself we are not opening up the stricture. We cannot open a scarry annular stricture, but we can open a kink or straighten a stenosis when we introduce the bronchoscope; I think we may sometimes have a stenosis which the bronchoscope fails to demonstrate.

I think, Dr. Eggers, that it would be of interest if our guests, Dr. Murstad and Dr. Giertz, would let us have a word or two on this subject.

DR. POL. N. CORYLLOS, New York, N. Y.—We have had the pleasure of hearing five excellent papers upon many problems of great interest to us all.

I agree with the remarks of Dr. Welles regarding the paper of Dr. Myers. When pneumothorax does not fulfill its purpose which is selective and efficient collapse of the diseased lobes, it is not only useless but dangerous. It exposes the patient and the physician to a feeling of false security that something is being done while the patient continues to spill bacilli around him and in his lungs; moreover, it exposes the patient to the complications of pneumothorax, namely, spontaneous pneumothorax and mixed infection tuberculous empyema. Therefore, pneumothorax should be completed when necessary by intrapleural pneumonolysis. I earnestly believe that pneumothorax cannot be successfully carried out in an institution unless skillful pneumonolysis can be carried out at the same place.

We have all gone through the difficulties of Dr. Freedlander and Dr. Wolpaw in trying to persuade our patients to accept thoracoplasty. We have had to overcome not only the ignorance of the patients, but often the stubborn objections of attending physicians. Fortunately, the good results obtained in thoracoplasty in our institutions have brought about the patients' giving their consents readily. Often the patient requests operation. It is pathetic to have to refuse to operate upon hopeless patients when they beg us to operate. It is not the same with private patients who are prejudiced against operation and who are often not encouraged by their physicians. This opposition also gradually disappears, however, as the number of successfully operated private patients increases by leaps and bounds.

*Dr. Lilienthal then spoke on apicolytic thoracoplasty with conservation of the first rib. This paper, read by title, he expects will be published in the Journal for perusal by this body.

Dr. Tuttle has developed in his paper the very important point of "tuberculin shock." Gatto (1931) has shown that intravenous injection of tuberculin in tuberculous children may cause shock with a marked decrease of globular resistance, hemolysis and considerable increase of bilirubin in the blood. In these cases, transfusion may cause rapid death. I have been interested in this question because of three deaths during transfusion in postoperative cases which developed unexplained shock.

I do not think that this complication, however, is as frequent as Dr. Tuttle mentions. In my experience, they have been extremely rare. Careful investigation has convinced me that in the majority of postoperative complications of thoracoplasty, we deal with hemorespiratory complications due to the mechanical or inflammatory decrease of the respiratory field, and death is caused by acute anoxemia. I have presented lately a study of my cases in a paper published in the *Quarterly Review of Sea View Hospital* (April, 1936). Immunology of tuberculosis is still in its infancy. We know nothing about immunity of tuberculosis and even less about its relation to allergy. We should, therefore, be very careful in drawing general conclusions. I am far from being convinced that desensitization with tuberculin or killed tubercle bacilli is of any real assistance in preventing postoperative complications. On the other hand, I do not know that bacteremia does not occur following thoracoplasty. Dr. Seigal, of the Pediatric Department of Sea View Hospital, was not able to find a single positive blood culture in a considerable number of postoperative cases followed for over two weeks after operation.

DR. J. A. MYERS, Minneapolis, Minn.—Our viewpoint regarding the treatment of tuberculosis is rapidly changing, because our methods of detecting this disease in the minimal stage have been fairly well standardized. Not long ago, approximately 80 per cent of the persons found to have clinical pulmonary tuberculosis had the disease in the moderately or the far advanced stage. The majority of these persons were definitely ill, in fact, the presence of symptoms was the chief reason for their first examination, and, of course, they required strict bed rest in addition to any collapse therapy that might be indicated. In recent years, however, health education programs have convinced many persons of the value of periodic examinations before illness appears. These programs have also resulted in numerous case-finding surveys. Moreover, for the past fifteen years, longitudinal studies of tuberculosis have been made; that is, groups of girls and boys have been closely observed that long to determine, so far as possible in the living body, the actual course taken by tuberculosis. These periodic examinations, surveys and longitudinal studies have resulted in striking revelations regarding tuberculosis. For example, we have learned that often the chronic reinfection form of pulmonary tuberculosis has reached such proportions as to cast a definitely visible shadow on the x-ray film; there is still an average period of approximately two to three years before symptoms are present to such a degree as to bring the individual to the physician for examination. It is during this long presymptom period when treatment is of most avail.

The work of Turner and Collins at Chicago has been very interesting with reference to the use of artificial pneumothorax and bed rest in minimal cases of pulmonary tuberculosis. In a good-sized group of patients with minimal disease treated by strict bed rest without collapse therapy, it has been shown that as many as 35 per cent showed progression of their disease. Hilleboe has pointed out that among persons discharged from sanatoriums, those with minimal tuberculosis have the risk of dying increased approximately four times, those with moderately advanced disease sixteen times, and those with far advanced disease forty times, over that of persons in the general population from which the patients were drawn.

I did not intend to leave the impression that all of the patients treated by ambulatory pneumothorax were on a compromise basis. In the early years of this work, ambulatory artificial pneumothorax was often begun as a compromise, but as time passed, such excellent results were obtained for many patients and the reports of others have been so satisfactory that we came to the conclusion that in properly selected cases ambulatory artificial pneumothorax is adequate, and there is now no hesitancy in recommending it for properly selected cases. This recommendation is made entirely on the basis of results obtained to date.

DR. S. O. FREEDLANDER, Cleveland, Ohio.—Just a word regarding the large number of refusals. In the population of our sanatoriums there are many foreigners, and the language difficulties in explaining what we are trying to do are great. Furthermore, at the time when most of these cases were recorded, we were somewhat undermanned, and we did not try to urge the operation too strongly. In the last few years, we have had very few refusals.

DR. WM. M. TUTTLE, Detroit, Mich.—I believe that Dr. Lilienthal has answered the question put by Dr. Welles with regard to the higher mortality after the second-stage operation. We have also found that the best way to avoid trouble after the second-stage is to do less at that time. When one considers the size of the collapse obtained when an extensive second-stage operation is added to a first-stage in which the first three ribs in their entirety have been removed, and that this collapse takes place overlying that part of the lung which contains the greater share of the disease, it will be readily seen that the amount of pressure with subsequent expression of tuberculoprotein may be large. We have learned, therefore, in the patient whose tuberculosis is relatively recent and whose sensitivity to tuberculin is high, to remove fewer ribs at the second stage. Generally, in such cases two ribs are removed, but at times only one rib is taken.

I think that the testing has been of distinct value in giving us some preoperative knowledge of the patient's probable postoperative behavior. When the disease within the lung appears recent, but the sensitivity to tuberculin is low, one may, with relative safety, proceed in the routine manner; on the other hand, the state of the disease process being the same, but tuberculin sensitiveness high, care must be exercised.

Of course, the ideal situation would be a desensitized patient, but as yet no method of rapid desensitization, which will at the same time preserve the patient's well-being, has been forthcoming. At present a period of months would be required to accomplish a satisfactory desensitization, and in the positive sputum case, this is too long.

Once the reaction is present we have felt that frequent small intravenous clyses of glucose help. It is also essential that if any signs of cyanosis appear, the patient be placed in an oxygen tent at once.

In answer to Dr. Coryllos' statement concerning the suppression of circulation in the collapsed lung, it is granted that such a suppression of lymph and blood flow occurs. This is probably an exceptionally fortunate turn of affairs for the patient. If no such suppression took place, the percentage of severe reactions would undoubtedly be much higher. I do not believe, however, that the suppression is ever complete.

With regard to the second point raised by Dr. Coryllos, it is granted that pneumonias on an allergic basis do occur; however, the type of reaction is different. It seems that it would be impossible to obtain the clear-cut perifocal type of reaction which we have shown by the insufflation of tuberculoprotein, for when tuberculin is placed within the lungs of previously infected animals, the resultant pneumonia is not confined to those areas bordering on old tuberculous foci. The inflammatory

lesions in such cases are widespread and the septal cell reaction, with subsequent outpouring of these cells into the alveoli, occurs wherever the tuberculoprotein comes in contact with lung tissue. The perifocal reaction is seen only when the tuberculin is given intravenously or subcutaneously. Furthermore, the pneumonia which results from the aspiration of substances to which the host is hypersensitive, has early signs of pulmonary consolidation, while in the type of reaction here described, the pulmonary manifestations come late or may be entirely absent.

DR. PAUL C. SAMSON, Ann Arbor, Mich.—In answer to Dr. Graham's question concerning atelectasis, I did not mean to imply that it was a favorable or an unfavorable phenomenon. Intermittent atelectasis is one symptom which I think is rather typical of recurring obstruction in these cases, probably due to tuberculous mucosal disease.

I agree with Dr. Eloesser that bronchoscopy is not final in diagnosis, but I think it is one of our valuable means of determining the condition inside the bronchi. I think, perhaps, bronchial kinks, and so forth, do develop following thoracoplasty, but in half of our cases in which tuberculous tracheobronchitis was found, it was discovered before any type of collapse therapy was given.

DR. JEROME HEAD, Chicago, Ill.—I have no further comments to make concerning my subject. I do wish to discuss Dr. Myers' paper. It is a subject in which I am deeply interested. Those of us who for years have urged the wider and earlier use of collapse measures cannot but be gratified at the present trends in the treatment of pulmonary tuberculosis. Dr. Myers has said and demonstrated that ambulatory artificial pneumothorax gives good results in "selected cases." I think that a great deal of emphasis should be put on the word "selected." There is great danger that selection will not be good, and that serious abuses are in the offing.

In regard to pneumothorax in general, I think that we should bear in mind that the entries are not all on the credit side. Over the past few years I have become increasingly convinced that a cavity held firmly open by adhesions was less likely to close than had pneumothorax not been used, and the treatment been limited to bed rest and paralysis of the diaphragm. If this is true, artificial pneumothorax is rendering stubborn and chronic many cases which might have healed readily had it not been used. At the Edward Sanatorium, we have observed that cavities thus frozen and stationary for many months under imperfect pneumothorax, frequently close rapidly if the pneumothorax is discontinued and a phrenicectomy performed. If pneumolysis is impracticable, this change of horses is always tried before thoracoplasty is resorted to.

AMERICAN BOARD OF SURGERY ORGANIZED

In answer to the widespread demand for an agency which will attempt to certify competent surgeons, the American Board of Surgery has recently been organized. This Board is a member of the Advisory Board of Medical Specialties, which includes all of the boards of certification for the different medical specialties which have already been organized. Since boards were in existence for the certification of practitioners of some of the surgical specialties, such as ophthalmology, otolaryngology, obstetrics and gynecology, genitourinary surgery and orthopedic surgery, it is expected that the American Board of Surgery will be responsible for the certification of general surgeons as well as those practicing in the remaining specialized subdivisions of surgery.

Acting upon the invitation of the American Surgical Association, the following surgical societies cooperated in the creation of the American Board of Surgery: The American Surgical Association, the Surgical Section of the American Medical Association, the American College of Surgeons, the Southern Surgical Association, the Western Surgical Association, the Pacific Coast Surgical Association, and the New England Surgical Society. The first three of these bodies, which are national in scope, have three representatives on the Board. All of the other societies have one representative each. The representatives of the cooperating societies are nominated by the society which they represent, and upon approval of the Board shall become members of it. The term of membership on the Board will be six years. The following were chosen to represent the cooperating surgical societies:

Dr. Evarts A. Graham Dr. Arthur W. Elting Dr. Allen O. Whipple	American Surgical Association.
Dr. Donald Guthrie Dr. Erwin R. Schmidt Dr. Harvey B. Stone	American College of Surgeons.
Dr. Fred W. Rankin Dr. Howard M. Clute Dr. J. Stewart Rodman	Surgical Section of the A. M. A.
Dr. Philemon E. Truesdale	New England Surgical Society.
Dr. Thomas Orr	Western Surgical Association.
Dr. Robert Payne	Southern Surgical Association.
Dr. Thomas Joyce	Pacific Coast Surgical Association.

The following officers were elected: Chairman, Dr. Evarts A. Graham; Vice Chairman, Dr. Allen O. Whipple; Secretary-Treasurer, Dr. J. Stewart Rodman.

Two groups of candidates are recognized for qualification by the Board: (a) Those who have already amply demonstrated their fitness as trained specialists in surgery; (b) Those who, having met the general and special requirements exacted by the Board, successfully pass its qualifying examination.

The first of these groups, the Founders Group, upon invitation by the Board will be chosen from the following: (1) Professors and Associate Professors of Surgery in approved medical schools in the United States and Canada; (2) Those who for fifteen years prior to the Board's organization have limited their practice to surgery; (3) Members of the American Surgical Association, the Southern Surgical Association, the Western Surgical Association, the Pacific Coast Surgical Association, and the New England Surgical Society, who were in good standing January 9, 1937.

All applications for the Founders Group must be received within two years of the Board's organization, January 9, 1937. No candidates for the Founders Group will be considered after that date.

Requirements for those to be qualified by examination will be as follows: (1) Graduation from a medical school of the United States or Canada recognized by the Council on Medical Education and Hospitals of the A. M. A., or graduation from an approved foreign school; (2) Completion of an interneship of not less than one year in a hospital approved by the same Council, or its equivalent in the opinion of the Board; (3) *Special Training:* A further period of graduate work of not less than three years devoted to surgery taken in a recognized graduate school of medicine, or in a hospital, or under the sponsorship accredited by the American Board of Surgery for the training of surgeons. This period of special training shall be of such character that the relation of the basic sciences of anatomy, physiology, pathology, bacteriology, and biochemistry is emphasized. Knowledge of these sciences as applied to clinical surgery will be required in the examination. Adequate operative experience in which the candidate has assumed the whole responsibility will be required. An additional period of not less than two years of study or practice in surgery; (4) The candidate must present to the Board sufficient evidence of good moral character as to justify the belief that he will not engage in fee splitting and other dishonest practices.

It is expected that the Board, with the assistance and cooperation of the American Medical Association and the American College of Surgeons, will be able to increase the facilities which now exist for the adequate training of young surgeons by means of residencies, fellowships, etc., in suitable hospitals.

The above requirements, especially those referring to surgical training, are subject to change from time to time as the existing opportunities for the training in this field of specialization may be broadened.

The qualifying examination will be divided into two parts: Part I, written; and Part II, clinical, bedside, and practical. The written part, Part I, will concern itself with general surgical problems and with the clinical application of the basic sciences of surgery to these problems. This examination will cover a period of three hours each and will be held simultaneously in as many centers as are necessary to accommodate the number of applicants who are eligible. Part II is entirely oral and will also concern itself, in the main, with general surgery, and, as stated for Part I, clinical application of the basic sciences to the clinical problem represented. In addition to this, in Part II, an examination will be given to test the candidate's knowledge of operative surgery, x-ray plate interpretation, and the principles and application of surgical anesthesia. This examination will be held in as many centers as the Board may determine necessary to accommodate the eligible candidates. Reexamination will be allowed, providing one year shall elapse between examinations.

The fee for Group A, the Founders Group, shall be $25. The fee for Group B shall be $75, payable as follows: $5 for registration, which shall be returned if the candidate is not accepted for examination; $20 for Part I; and $50 for Part II. The same fee will be required for each reexamination. Once the candidate has become qualified, he will have no further financial obligation to the Board.

This Board is a nonprofit organization. All fees will be used, after a reasonable amount is set aside for necessary expenses in maintaining its office, conducting examinations, etc., to aid in improving existing opportunities for the training of the surgeon.

A certificate attesting to a candidate's qualifications in surgery after meeting the requirements of the Board will be issued, having been signed by its officers.

Any certificate issued by the Board shall be subject to revocation by the Board at any time in case it shall determine, in its sole judgment, that a candidate, who has received a certificate, either was not properly qualified to receive it or has become disqualified since its receipt.

The Board will hold its first examination (Part I, written) on September 20, 1937. All inquiries concerning applications for this examination should be received by the secretary's office promptly.

Requests for booklets of information, application blanks, and other information should be addressed to the Secretary, Dr. J. Stewart Rodman, 225 South 15th Street, Philadelphia, Pennsylvania.